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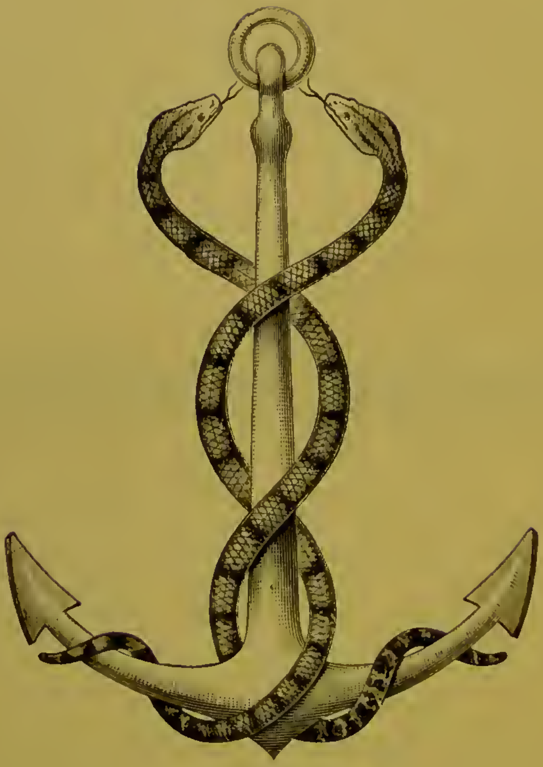

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TEXT-BOOK
OF
MEDICINE

EDITED BY

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VOLUME SECOND

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SECTION V.

HÆMOPOIETIC SYSTEM.

DISEASES OF THE BLOOD.

INTRODUCTORY.

The blood, including the lymph, differs from the other tissues, in that it comes into close contact with all parts of the body, and any alteration in it necessarily affects the nutrition and well-being of the whole organism. It serves as the vehicle for carrying numerous constituents necessary for life, as well as the waste products resulting from cell activity. Within certain limits, therefore, its composition is very variable, but in health these limits are remarkably narrow, and hence the composition of the blood may be said to remain practically constant. This is brought about, partly by the close relationship between the blood and the lymph, enabling the former to draw on the latter continuously or in emergency for fluid and albuminous constituents, by the regularity with which digestion proceeds in health, and by the constant regular activity of the excretory glands and surfaces. Interference with these last may produce rapid death, as in non-excretion of carbonic acid gas; or slower poisoning, as in uræmia. Alterations in the functions of any important organ may also considerably affect the blood, as in jaundice or acetonæmia.

Amount.—Vierordt and other physiologists have fixed the total amount of blood in the body as about one-thirteenth of the body weight. Their conclusions have necessarily been drawn from observations on animals, and, apart from inexactitude in the methods of determination, the result can only be regarded as approximate, both as an average and for the individual. There are probably very considerable differences in the vascular capacity of different people, while the general condition of the body, especially with regard to muscular and fatty development, undoubtedly influences it in any one person, and at different times. Oertel's clinical observations on cases of cardiac disease point to the conclusion that the total volume of the blood may be made to vary within very considerable limits by regimen and treatment; Bollinger states that habitual excess in beer drinking may lead to an increase in the total volume; and it is known to be lessened in cholera and diabetes, and increased in dropsy. We may, however, accept one-eleventh to one-fourteenth of the body weight as an approach to accuracy in the average individual. A loss of 30 to 40 per cent. of its volume will cause death if it be sudden, but gradual loss to a much greater extent can be borne.

Specific gravity.—This varies according to the amount of solids present in proportion to the water. It may be estimated by weighing, and, if capillary tubes are used, only a very small amount of blood may be necessary to carry out the determination; clinically, however, it is usually determined by dropping blood into fluids of known specific gravity, with which the blood will not mix (chloroform and benzol, for example). When the drop rests midway between top and bottom of the fluid, the two are equal in density. The specific gravity of the blood is slightly less in women and children than in adult men, and various physiological conditions modify it, but only to a trifling extent. In disease it is influenced chiefly by the number and condition of the red blood corpuscles, but also by the amount of water. It is diminished in anæmia and dropsy, and increased in conditions which drain off the watery constituents. Grawitz states that it may be temporarily lessened by administering nitrite of amyl, or by any procedure which dilates the blood vessels.

Composition.—The blood consists of an albuminous plasma, which holds in solution salts, oxygen, nitrogen, and carbonic acid, along with small quantities of a large number of food and waste materials, the formed elements being the red and white corpuscles and blood plates. Its reaction is alkaline. Its average specific gravity is about 1055 to 1060, but it may vary in health from 1045 to 1075, or even within wider limits. When dried the solid residue is about 22 per cent. The total volume of red corpuscles in relation to the whole blood varies a good deal, but the average is somewhere about 45 per cent. The function of the plasma is, in general terms, nutritive, while the red corpuscles carry oxygen to the tissues. Possibly these may have other functions, as, in lead and other metallic poisoning, they often contain a certain quantity of the metal.

Plasma.—The plasma is yellowish coloured, alkaline, and tends to clot rapidly after being withdrawn from the vessels. It contains various proteids—fibrinogen, serum globulin, serum albumins (one or more), and nucleoproteids—fatty bodies, sugar and other carbohydrates and salts. The serum has an average specific gravity of 1028 to 1030, and contains about 10 per cent. of solids. The salts are chiefly sodium chloride, along with magnesium, calcium, and potassium compounds. The richness of the blood in proteids is lessened by severe hæmorrhage, or by any chronic drain on the system, such as albuminuria or suppuration, but if appetite and digestion remain good, it may be a very long time before the latter make any marked impression. Good or excessive feeding does not increase the proteids beyond a certain physiological maximum.

The alkalinity of the blood is due to alkaline carbonates chiefly, and is reckoned as equal to about 0·2 to 0·3 NaHO per 100 grms. The methods used for its estimation are not very exact, and it is a changing quantity, but only within very narrow limits, as an alkaline reaction of the blood plasma and lymph is necessary for the maintenance of life. There is a constant setting free in the tissues of acid waste products—carbonic, lactic, and fatty acids—which require to be neutralised. This demand is met in man and in the carnivora by continuous production of ammonia in the system. By this means the alkalinity of the blood is kept practically constant, and hence the giving of acids scarcely, if at all, lessens it, or affects the reaction of the urine. In men the blood is stated to be normally more alkaline than in women and children, while the alkalinity suffers a diminution during muscular activity, in tetanus, strychnine poisoning, in fevers, and in diabetic coma. The administration of alkalies has little or only a transient effect in increasing the alkalinity, owing to the rapidity of their excretion.

Red corpuscles.—These are yellowish coloured, biconcave discs, about 1·5 μ in thickness, and varying a good deal in diameter. Hayem classes 75 per cent. as medium (7 to 8 μ), 12 per cent. as small (6 μ), and 12 per cent. as large (9 μ), from which it is evident that the medium-sized are in very great preponderance. Age and sex have practically no effect on the size, except in the newly-born, where there is a number of very small and large forms. They are generally described as consisting of a stroma, in which lie the protoplasm and colouring matter ; but Foà states that they are made up of an outer layer of hæmoglobin, inside which is found a meshwork containing granules, and under this is the homogeneous cell protoplasm. Their specific gravity is about 1080 to 1085, so that they are very considerably heavier than the plasma, and can scarcely be said to "float" in it, although this expression is often used.

The enumeration of the red corpuscles was first carried out by Vierordt (1852), who used a saline solution as a diluent. Since then numerous improved forms of hæmacytometer have been invented, and with care the error in counting need not be more than 3 or 4 per cent. above or below. These errors are due to the construction of the instrument, and to temporary alterations in the fluidity of the blood.

They number about 5 millions per cubic mm. in men, and about 4½ millions per cubic mm. in women. The number cannot be increased permanently in health, iron and a liberal diet having no effect unless the corpuscles have been previously deficient. Even when blood is injected into the circulation, the excess of corpuscles has entirely disappeared in three weeks or less. There is no doubt, however, that habitual generous living, as contrasted with the reverse, tends to a general richness of all blood constituents. It is often stated that there is an increase immediately after meals, but this is not the case. With the present methods of counting, the effect of menstruation on the number is rarely visible, nor in healthy women does lactation diminish the number. Regnard, Miescher, Viault, and others have shown that living at a high altitude greatly increases the number. In Lima, Viault found that his corpuscles numbered 5 millions per cubic mm., but after three weeks' residence in the Andes they had risen to 8 millions, and it has been found that dwellers in rarefied atmospheres have normally this excess. Schauman and Rosenqvst have proved that the rarefaction of the air is the only factor of high altitudes in determining the increase. On returning to the sea level the number rapidly diminishes again. Pathologically, the number may be greatly diminished in different forms of anæmia—after bleeding, in malarial disease, lead poisoning, fevers, and many other conditions. The lowest limit usually consistent with life is between 300,000 and 400,000 per cubic mm., but lower counts have been often recorded. Any permanent increase in their number (plethora) is rare, but is found in cyanosis, especially that due to congenital heart disease, where they may reach 9 millions per cubic mm.

In anæmia, the corpuscles may be very considerably altered, both in size and shape, very small forms (2 μ), or very large (12 to 14 μ), being often seen. They may be pear-shaped, ovoid, or otherwise distorted, and this condition has been called poikilocytosis, and the misshapen corpuscles poikilocytes. Their resistance to destructive agencies may be greatly lessened, and under the microscope they frequently do not form good rouleaux.

Nucleated red corpuscles.—These are found in the fœtus, in the newly-born, and in adults during severe anæmia. They are not normally present in health, and their presence accompanies rapid regeneration of the blood. They vary a good deal in size, some of them being very large.

Microcytes is a term which has been applied to very small coloured corpuscles. They are found both in health and disease, but are especially numerous in anæmia. They are said to be portions of broken-down hæmocytes, but it is possible they may be very small abortive corpuscles.

Hæmoglobin.—The hæmoglobin forms about 14 per cent. of the whole blood. In composition it is a proteid, and on splitting yields about 96 per cent. albumin, and 4 per cent. hæmochrome, an iron-containing body. Its iron is derived from the organic iron of the food which is absorbed and stored in the liver cells, from which it is conveyed to the bone marrow as an albuminous compound, but we are ignorant of the details of the process. The amount of hæmoglobin may be greatly diminished in anæmia, as also the hæmoglobin value of individual corpuscles. Clinically its amount is estimated by diluting a small measured quantity of blood, and comparing it with a standard colour. This method is not particularly exact, but for details the reader must be referred to works on clinical methods.

Leucocytes.—The white blood corpuscles vary in appearance, and present morphologically three chief types—(1) Mononuclear round cells. These are small and large lymphocytes with homogeneous protoplasm and a deeply staining nucleus. (2) Large cells with finely granular protoplasm, staining very readily. In these the nuclei are either large and of a bent or horse-shoe shape, or may be divided up into several smaller nuclei, which may or may not be connected together by narrow filaments (polynuclear forms). (3) Large cells with coarsely granular protoplasm, staining deeply with acid stains (eosinophile cells). Ehrlich, Kanthack, and others have classified them in a much more detailed manner, according chiefly to the appearance of the nuclei and their affinity for different aniline stains; but the above classification seems to me sufficiently complex, considering how opinions differ regarding their clinical significance. Some writers, such as Gulland, hold that all forms are derived from the lymphocytes, while others are of opinion that they differ in origin. If the former opinion be correct, as I think it is, then the more detailed classifications are not necessary. Statements have been frequently made as to the relative proportions of the different kinds, but this is a very changing quantity, as they are constantly passing from blood to connective tissue, and back again. Their distribution in arteries, veins, and capillaries seems to vary greatly at different times, but they are said to be more numerous in the last. Their number in any specimen of blood is therefore variable, as is also their relative proportion to the red corpuscles. The former varies from about 4000 to 10,000 per cubic mm., and the latter from 1 to 500 to 1 to 1200. They vary in size from 7 μ to 10 to 12 μ. White cells appear in the blood only some days after birth; they are formed in the various lymphoid tissues, in which they also undergo development into the different forms. Their number is greatly increased by the taking of food (owing to their rapid mitotic reproduction in intestinal tissue and glands), in certain fevers, in pregnancy, and by certain drugs, while it is diminished by fasting. They are counted in much the same method as the red corpuscles.

Hæmatoblasts or blood plates.—These have been very fully described by Hayem (1877), and by Bizzozero (1882). They are small, round, or ovoid bodies, homogeneous or finely granular, and contain no hæmoglobin. They average about 2 to 3 μ in diameter, and are faintly tinged by alkaline stains. To see them well they should be rapidly examined in freshly drawn blood, as they are very sticky, and run together in heaps, or lie against the red or white corpuscles. Their physiological rôle is not known, but they are supposed to have some connection with coagulation, and with the formation of thrombi. Hayem regards them as the precursors of the red corpuscles. They have been seen in the vessels during life, and their number has been variously estimated as from 180,000 to 500,000 per cubic mm. They are difficult to count accurately, and their number probably varies in different samples of blood, even from the same individual. They become more numerous during recovery from severe anæmia, and less numerous under the action of blood poisons.

Blood formation.—In early uterine life the red corpuscles seem to be formed in the walls of the vessels. Later, the liver, spleen, and bone marrow take up this function. After birth, the red marrow is the chief if not the only organ in which they are formed, but there is great difference of opinion as to this and regarding the exact steps of the process. Some authors hold that the spleen and lymph glands are also blood-forming organs, but the evidence adduced cannot be considered very convincing, and certainly, in pathological states of the blood, it is chiefly in the bone marrow that active regenerative changes are seen.

Blood destruction.—Most physiologists hold that an active destruction of hæmocytes is constantly taking place in the liver, spleen, and bone marrow, the colouring matter being excreted as bile, urinary, and other pigments, while the iron is retained largely in the body and used over again. Quincke holds that the average life of a red corpuscle is three weeks, and that consequently the whole of the red cells are renewed once in that time; under the name of siderosis he has described deposits of iron in different organs, resulting from this breaking down. The evidence in favour of the occurrence of such wholesale destruction has never seemed to me quite convincing. It is assumed that once the red cells are launched into the circulation, no wear or regeneration takes place in them as in other tissues, and that they play a purely passive part as regards their own metabolism. The life history of individual corpuscles has never yet been followed out, and hence definite knowledge is lacking, but the following view is permissible also by the present state of our knowledge. A certain number of corpuscles are continually wearing out as it were, rather than being actually destroyed, and these are dealt with by leucocytes in the spleen and liver, but that any more active destruction takes place by the cells of these viscera seems to me highly improbable. This view is borne out by certain observations of Minkowski and Naunyn. After poisoning animals with arseniuretted hydrogen, they observed in the liver and bone-marrow large numbers of leucocytes carrying partially broken-down red corpuscles, whereas elsewhere such leucocytes were rare; the explanation of this is that slowing of the blood stream is necessary to allow the white to deal with the effete or weakened red corpuscles; and hence, although the breaking down occurs chiefly where the circulation is very slow (as in the liver or spleen), it occurs not through the agency of those organs but by means of the leucocytes. I believe it is highly probable that normally the process is somewhat similar.

Regeneration of the blood.—After a profuse hæmorrhage there rapidly occurs an increase of the white corpuscles, probably from the lymph streaming in to supply the lost fluid. The red cells also soon begin to increase rapidly in number, and under good conditions may reach their former total in three or four weeks, but they are deficient in hæmoglobin, and many of them are ill-formed, often small, or large, or nucleated. The increase in the hæmoglobin is always very slow, and even when iron is given it lags greatly behind the increase in the corpuscles. Coincidentally there is an increase in the activity of the red marrow, which becomes larger in amount, deepens in colour, and becomes soft and lymphoid in consistence, while many large nucleated red cells are observed in it. The spleen is said by some observers to participate in this activity.

Melanæmia.—In old and severe malarial cases, especially after an exacerbation, small dark particles of altered blood pigment are found in the circulation, but more especially are stored in the liver, spleen, bone marrow, kidney, and brain. Frequently these particles are also seen in the leucocytes. It is said that this black substance (melanin) is formed by the destruction of red corpuscles in the liver and spleen, but more probably they disintegrate in the blood stream under the action of malarial organisms, the granules being taken up by the leucocytes, carried to the various organs, and there deposited. In animals, after transfusion of blood, similar changes occur from the breaking down of the excess of hæmocytes.

ANÆMIA.

Anæmia (want of blood), or oligæmia (deficiency of blood), are terms which might literally be applied to denote a deficiency in all the blood constituents; but, practically speaking, when we use the word anæmia it is understood to imply deficiency either in the amount of hæmoglobin, or in the number of hæmocytes, or in both. It is comparatively rare that the constituents of the blood plasma have been found to be deficient, and, as a matter of fact, so few observations have been made on its composition in disease, that we have little clinical knowledge regarding it. On the other hand, a great deal of information has been collected regarding the condition and number of the red corpuscles. If precision of nomenclature were generally adopted, and a distinction habitually made by practical physicians according to the elements of the blood which are deficient, the following terms would doubtless be more used than they are at present:—*Oligocythæmia*, a decrease in the number of red corpuscles; *oligochromæmia*, a deficiency in the hæmoglobin; *hydræmia* and *hypalbuminosis*, an increase in the water, and a decrease in the albuminous constituents of the plasma respectively; while *spanæmia* is a term signifying poor or thin blood generally. It is found, however, that these conditions are often conjoined, and one comes across the terms chiefly in systematic treatises.

Anæmia is the accompaniment of a very large number of serious diseases, such as chronic renal disease, acute and chronic febrile conditions, malaria, advanced carcinoma, syphilis, lead and mercurial poisoning, etc. The stage of the disease makes a great difference, however, as regards the actual blood condition, which is sometimes a mere subsidiary symptom, and at other times a very prominent feature. Bad hygienic surroundings of all kinds, just as they lead to a poor condition of the general health, induce at the same time a certain poorness in the blood; deficient development of the arterial system (Virchow), constitutional and hereditary influences, a single or repeated loss of blood, may all lead to severe anæmia. In all these cases there is probably a disproportion between the consumption and renewal of the corpuscles. Disease of the bone marrow, by impairing blood formation, leukæmia, and many other conditions, may also seriously interfere with the composition of the blood.

It has been customary to divide cases of anæmia into two classes—(1) secondary, and (2) primary or idiopathic; the former including all those cases enumerated above, where the blood is affected obviously as a consequence of some other condition; and the latter including especially chlorosis, pernicious anæmia, and leukæmia, where the cause is often undetermined. This latter class, however, has gradually diminished as our knowledge has increased, and such a classification can only be regarded as provisional. It should be now dropped altogether, because in many of these cases the cause of the anæmia is certainly known, and most probably in none of them does it primarily take its rise in the blood itself.

ACUTE ANÆMIA.

This term has been applied to rapid loss of blood, occurring either externally or internally, and extending over a few minutes, a few hours, or two or three days.

Etiology and symptoms.—It may occur in the healthy from injury to large vessels, or in the course of aneurysm, phthisis, gastric ulcer, parturition, etc. Its treatment and prognosis are therefore very variable, and can scarcely be discussed in detail in this connection. The symptoms are the well-known ones of acute hæmorrhage, their severity depending largely on the amount of blood lost. There occur pallor, collapse, coldness of the skin, with cold perspiration; the heart is quick; the pulse small, compressible, and excitable; the blood pressure falls rapidly, severe fainting may ensue, and there is marked muscular weakness. The patient often shows a good deal of mental excitement, with anxiety and restlessness, and the anæmia of the brain leads to disturbance of special senses, shown by symptoms such as flashes before the eyes, dimness of vision, and singing in the ears. Death may occur in convulsions or quietly, and in a few minutes or after some days. If the patient survive, great thirst is experienced, along with general muscular and cardiac weakness, feeble digestion, nervous irritability, sleeplessness, and other symptoms of impaired nutrition in all the functions and systems of the body. In the comparatively healthy the fluids and albuminous matters are soon made good from the lymph and from the food, the withdrawal of the former from the tissues being the occasion of the severe after-thirst. The red blood corpuscles regenerate very rapidly, and even after severe bleeding may regain their normal number in about a month, although many of the newly-formed hæmocytes are small, or large, or slightly misshapen, and are more or less deficient in hæmoglobin. The hæmoglobin, even under favourable conditions, takes three or four months, or longer, to reach its normal standard. The regeneration of the blood is much influenced, however, by the general condition of the patient.

Treatment.—The immediate treatment depends largely on the cause, and may be purely surgical or obstetric in character. If the patient survive the acute hæmorrhage, he should be kept strictly in bed, in a cool room, with few bed-clothes, and with the head low. Even slight exertion should be avoided for many days. Opiates often quiet the restlessness, tend to control mental excitement, and lessen the chance of recurrence. The food should be cold and fluid. The use of other measures must be regulated by the site of the bleeding, and the general nature of the case. Ergot is useful if the bleeding be from the uterus, or from a small artery, but most of the other so-called hæmostatic astringents are of very doubtful value. If it be considered necessary to replace the loss of fluid, defibrinated human blood may be transfused into a vein, mixed with phosphate of sodium (sp. gr. about 1030), or one or two pints of sodium chloride ($\frac{3}{4}$ per cent.) may be given slowly per venam, or subcutaneously, or by the rectum.

The subcutaneous injection of defibrinated human blood is advocated by von Ziemssen, but in my own experience this method is inferior to the others.

CHLOROSIS.

This disease, commonly known as the "green sickness," is a chronic morbid condition, arising during the years of puberty and adolescence, practically confined to the female sex, and characterised by a diminution in the amount of hæmoglobin, with or without a deficiency in number of the red blood corpuscles, most of the other symptoms being directly traceable to the blood change.

History.—Chlorosis was known to Hippocrates, and has been recognised by most writers, from the earliest to the present times. Certain authors have regarded it as due to defective function, or developmental faults in the reproductive system; others, as the expression of a special cachexia, or as a disease arising in different parts of the nervous system, as secondary to all kinds of digestive troubles, and to other causes. Willis (seventeenth century), Boerhaave and Cullen (eighteenth century) recognised the watery condition of the blood. Foedisch (1832) found a diminution of iron, and Andral and Gavarret (1840) a diminution in the number of red corpuscles. Duncan (1867) first pointed out that the most characteristic change in the blood is a deficiency of hæmoglobin, along with lessened specific gravity, and his observations have been confirmed and greatly extended by numerous subsequent observers. The name chlorosis was given to it by Jean Varandal (Montpellier, 1620), from χλωρος, yellowish green, owing to the tint of the skin often seen.

Etiology.—The view taken of the etiology of chlorosis will be influenced largely by the opinions held regarding its nature. Many authorities hold that it is primarily a blood deterioration; and if that be so, then it must be acknowledged that no one has as yet been able to fix definitely on a constant cause. On the other hand, if it be regarded simply as a condition of the blood secondary to numerous causes, and which condition leads to other changes in bodily chemistry and function, it will be admitted that in many cases one is able to distinctly indicate certain influences capable of bringing about the blood change. In few morbid states have so many or so conflicting opinions been put forward as to causation. Many of these, in the light of modern knowledge, are manifestly incorrect, and hence it will be necessary to refer only to those which are still current. Virchow held that chlorosis is due to congenital hypoplasia of the arterial system, but this view is contradicted by the fact that such a condition is permanent, whilst chlorosis is transient. It has also been attributed to deficient development of the reproductive system (Fränkel). Another view, which has obtained wide currency, is that chlorosis is due to constipation, with consequent formation of poisonous ptomaines in the alimentary canal, which are supposed to be absorbed and to break down the red corpuscles (Andrew Clark). Many cases, however, do not suffer from constipation, while accurate observations have proved that there is no excessive decomposition in the intestinal canal, and that fewer corpuscles break down than in health. Hannon and, later, Bunge, have advanced the hypothesis that there is intestinal sepsis, resulting in the excessive production of sulphides, which precipitate the iron from the food, and make it unabsorbable. This theory has also been disproved from many sides. Absence of light and fresh air, heated rooms, want of exercise, unhealthy excitement, depressing emotions, indigestion in various forms, insufficient food, and menstrual disturbances have all been advanced as causes. Lloyd Jones states that the blood changes seen in chlorosis are simply an exaggeration of what normally occurs at puberty in girls, but not in boys, and is of opinion that there is some unknown cause underlying not only the blood changes, but responsible also for many of the other symptoms. He fails to explain, however, why chlorosis should so often occur several years after puberty. Meinert, in an elaborate monograph, has attributed it to dilatation of the stomach, with displacement; von Hösslin, to repeated small unobserved bleedings from the bowel; while Hayem, von Noorden, and Birch-Hirschfeld, all regard it as due to weakness in the blood-forming organs. An objection which may be taken to this last view, is, that in many cases the corpuscles are normal in number, the deficiency being confined to the hæmoglobin.

Regarded broadly, it is plain that any deficiency in corpuscles and hæmoglobin must be due to excess in breaking down, to direct loss by bleeding, or to some defect in blood formation. In chlorosis it has been shown that the first of these causes is not present, the second is frequently present as normal or excessive menstrual loss, while the defective formation affects the hæmoglobin rather than the corpuscles, although the latter are most commonly subnormal in number. The most striking features of the incidence of chlorosis are the sex and age of the patients, and the explanation of its etiology must be looked for in the condition of the blood and nutrition found in adolescent females as contrasted with those found in males at the same age. My own observations have led me to conclude that the condition is secondary, and due chiefly to blood loss and to insufficient ingestion of iron in the food. It is infinitely more frequent in young women, because normally their blood is poorer, they ingest less iron as food, and they are subject to periodical blood loss. In women the blood contains about 10 per cent. fewer corpuscles, 8 to 10 per cent. less hæmoglobin, and about 5 per cent. more water than that of men at the same age. Further, this deficiency is most marked during the years when chlorosis is most common. The very rapid growth of girls at puberty throws a great strain on all the bodily functions, including blood making and digestion; the appetite is often extremely poor, and this affects not only the general nutrition, but also the amount of iron ingested. Whereas a normal dietary was found to contain daily from $\frac{1}{11}$ to $\frac{1}{8}$ gr. of iron, the dietary of three chlorotic girls only contained from $\frac{1}{50}$ to $\frac{1}{20}$ gr. A certain amount of iron is excreted daily, and if this loss be not made up by the food, the organism gradually becomes deficient in iron and hæmoglobin; also at each menstrual period about ½ to 1½ grs. of iron are further lost to the body, and in the anæmic there are no reserves in the liver or elsewhere to make good this loss. The amenorrhœa so frequently seen is therefore a beneficial and protective condition. Chlorosis undoubtedly comes on after menorrhagia, and in one case under my care it was due to chronic bleeding from a rectal polypus. There is probably a certain hereditary predisposition in some cases, as the members of certain families seem specially liable to it. If this view be correct, the causes of chlorosis ordinarily alleged may be regarded as purely predisposing, while the direct causes are—(1) Excessive menstrual loss (or other blood loss). This may be relative, that is, too much for a weakly or rapidly growing individual to bear, or it may be actually large. (2) Insufficient ingestion of iron as food. Anything which diminishes the appetite—as dyspepsia, constipation, want of exercise, unhealthy occupations, change from country to town life, etc.—all predispose to anæmia. In many cases there is a combination of these two causes.

Chlorosis occurring in boys and non-menstruated girls is explained by the strain thrown on the body by rapid growth, which tends to lessen appetite and blood formation. In the anæmia of the menopause, dyspepsia and menorrhagia sufficiently account for the deterioration of the blood; and gastric ulcer, where one has a combination of poor appetite and blood loss, often leads to very severe and obstinate chlorosis. Chlorosis, therefore, is most probably a secondary anæmia, predisposed to by many causes, but actually induced by deficiency of iron in the body. It is common among all classes, and in all civilised and semi-civilised countries.

Morbid anatomy and pathology.—The condition of the blood, as previously described, is the most important feature; in addition, during recovery, a few nucleated red cells may be seen. There have not been many post-mortem examinations of cases, and in the few which are recorded death has occurred from intercurrent disease, which affects the results somewhat. In one case Tissier found fatty degeneration of the liver, and the bile almost devoid of pigment. In a case of my own, where death occurred suddenly after violent hæmatemesis, there was extreme fatty degeneration of the heart muscle, and practically complete absence of iron from the liver, spleen, and other organs.

The fatty degeneration is due to want of oxygen, following on the lack of hæmoglobin, as the albuminous tissues disintegrate, and the products not being oxydised remain in the cells.

Symptoms.—The symptoms are very largely those of chronic anæmia, but they vary greatly in severity, this depending chiefly on the extent to which reduction in the amount of hæmoglobin has gone. The patient has usually an extreme pallor, although sometimes the cheeks flush readily, and the complexion varies somewhat, according to the texture of the skin; in blondes it may be a dull white or a transparent white, while in brunettes it has often a greenish tinge. The ears frequently present the peculiar semi-transparent appearance of old wax, and the veins on the back of the hands have a delicate violet tint. There is no tendency to emaciation, but rather the contrary. It is, in fact, frequently stated that chlorotic girls are markedly plump, but on the whole they present in this respect much the same appearance as their non-chlorotic companions. The mucous membranes of the mouth and eyes are also pale, and the vessels ill-defined. There is marked languor and dislike to sustained exertion, either bodily or mental, although under the influence of excitement a large amount of either kind of work may be accomplished.

On examining a drop of blood, withdrawn by a prick through the skin, it appears pale and watery to the naked eye; under the microscope it may or may not form good rouleaux, but many of the corpuscles are seen to be pale and sometimes slightly deformed in shape, while many are smaller than usual. In a fair proportion of cases their number is normal, in a large proportion they amount to between 2 and 4 millions per cubic mm., while less than 2 millions is decidedly rare. Hæmatoblasts are abundant. The hæmoglobin is always greatly reduced in amount, even falling to below 20 per cent. of the hæmoglobinometer scale, but from about 30 to 60 per cent. is commonly found. The hæmoglobin value of the individual corpuscles is always more or less reduced, as is also their oxygen-carrying capacity. The blood plasma is normal in composition.

In most cases the digestive functions are greatly interfered with, the appetite is poor and in some cases remarkably small, while dyspepsia of all degrees is met with. The free hydrochloric acid of the gastric juice may be increased, diminished, or normal in amount. Obstinate constipation is frequent, but by no means invariable, while there is sometimes a depraved appetite for such things as chalk, coal, and linen articles.

In all cases there is very marked breathlessness, especially on going upstairs or on exertion of any kind, but it is quite absent when the patient is at rest. The heart is said to be dilated, but this can very seldom be actually determined by physical examination, and probably is not constant. It is easily excited, palpitation and præcordial oppression are very common; the pulse is full and regular and sometimes a little rapid, but really presents no characteristic features. Hæmic murmurs of a soft blowing character are very commonly heard over all the cardiac areas. If present anywhere, they are always audible over the pulmonary area, and are usually most intense there, although sometimes loudest in the mitral area. The latter murmur is conducted into the axilla. No satisfactory explanation of the cause of these murmurs has ever been given, and for an account of the various views held regarding them the reader must be referred to works dealing specially with physical diagnosis. A loud humming murmur (*bruit de diable*), constantly varying in intensity, is also heard in the external jugular vein, especially on the right side of the neck; on palpating the vein a thrill can be felt, and both murmur and thrill may be made to disappear by compressing the vessel. Its origin is due to fluid veins, arising from the comparatively dilated state of the lower end of the jugular. It may be heard also in many healthy subjects, but is always more intense in the anæmic; a similar murmur is often audible over the eyeball and over the torcular Herophili. If the patient is going about, there is usually slight œdema at the ankles; and this during the night's rest is often transferred to the face, making the eyes especially swollen and puffy-looking. Fainting attacks or frequent feelings of faintness are very common. All these symptoms are due partly to the condition of the blood and the want of oxygen, and partly to that of the heart.

Disorders of menstruation are very often present. In rare instances the patient has never menstruated, sometimes the anæmia has been preceded by menorrhagia, more rarely it is accompanied by it. Scanty and irregular menstruation and amenorrhœa are much more frequent. Leucorrhœa is also common as an accompaniment of chlorosis, and often aggravates greatly the feeling of malaise.

The nervous system is usually below par and is easily fatigued; headaches, neuralgic pain in different viscera, infra-mammary and other neuralgias, are very common, while the vasomotor system is extremely irritable. The urine is pale and abundant, the solids, including the pigments and urea, are usually under the normal as a consequence of the lessened metabolism and appetite, but it presents no abnormal constituents.

Slight feverish attacks are by no means uncommon, and have been described chiefly by French writers as a special type—*la chlorose fébrile.* Their occurrence has been attributed to the blood condition, but possibly they are simply due to unimportant microbic infection, occurring in the course of the illness. Gastric ulcer is a common and very troublesome accompaniment of chlorosis, while the formation of thrombi in the veins, and the occurrence of optic neuritis are distinctly rare.

Diagnosis.—Although no one symptom is characteristic, yet in practice diagnosis is easy when the age, sex, bloodlessness, and general symptoms are taken into account. In the great majority of cases the first attack occurs between the ages of 16 and 21, after which it becomes rapidly rarer and is decidedly rare after 25. The anæmia of young girls in early tuberculosis has been often mistaken for it, but it is not nearly so amenable to treatment. In other anæmias the underlying cause can frequently be discovered, and they are uncommon at the ages when chlorosis is most frequent.

Prognosis.—Death never occurs as a direct result of chlorotic anæmia. Relapses are, however, very common and sometimes occur at intervals during several years. They can usually be avoided if the patient is careful to follow out the treatment recommended.

Treatment.—Clinical experience has proved that the most important remedial measure is the administration of iron. Whether given by the mouth, by the rectum or subcutaneously, it is absorbed and utilised for the formation of new red corpuscles and hæmoglobin. Although there has been much discussion regarding this, its direct absorption from the intestine has now been demonstrated (Macallum). It appears to be stored in the liver, and is there converted into an albuminous compound which is the predecessor of hæmoglobin. Its administration ought to be continued for eight or ten weeks at least, because comparatively little is absorbed, and the corpuscles which have been formed during the anæmic state are poor in hæmoglobin, they disappear slowly from the circulation, and are only gradually replaced by others rich in iron. The inorganic preparations in ordinary use are perfectly efficient, and, on the whole, give the best results. The choice of the particular preparation is guided to a very large extent by the condition of the stomach: if that organ be irritable, it is best to use the least irritating—such as reduced iron, ferrous carbonate in one form or another, or one of the scale preparations. The protochloride and sulphate are distinctly more irritating, and ferric salts still more so.

Large doses are often recommended, and are held to be most efficacious and rapid in action, probably because more iron becomes absorbed when it is present in large quantity in the bowel. Such large doses often interfere a good deal with digestion, and most cases recover perfectly well and quickly when 3 to 6 grs. of ferrous carbonate or reduced iron are given daily. Any preparation of iron should be given directly after meals, as it then interferes less with digestion. Constipation can be treated by rhubarb or salines, or any other mild purgative.

Arsenic, manganese, and some other metals, given along with the iron, have been supposed to increase its efficacy. Manganese and the others are certainly useless. Arsenic is supposed to stimulate the formation of the red corpuscles, and in this respect may be of some value; but when cases of chlorosis are treated by arsenic alone, no improvement takes place until sufficient doses of iron are added to it. Fatty and albuminous foods and cod-liver oil seem also to hasten blood formation. In very obstinate cases residence at high altitudes has been recommended; this acts as a stimulant to the blood formation, and it is said that its effect on the blood-forming organs is very often a permanent one. Dyes, Wilhelmi, and others have advocated repeated small phlebotomies as another means of stimulating these organs, and undoubtedly they appear to do so, but such a method is not suitable for ordinary practice.

As regards diet, no specific directions can be given, as it must be regulated by the condition of the stomach and digestive organs. When gastric digestion is fairly good, an ordinary mixed diet, with a reasonable amount of butcher meat, should be recommended; if there be severe dyspepsia or gastric ulcer, the diet must be regulated accordingly.

Good general surroundings, attention to personal hygiene, treatment of dyspepsia and of constipation, are of the utmost importance, as thereby the appetite becomes larger and the patient gets into the habit of taking a sufficient quantity of food. The same holds good of country air, spa treatment, hydrotherapy, and massage, all of which are useful adjuncts to more direct therapeutical measures. If the patient remain under the unfavourable conditions which predispose to the disease, treatment is often very unsatisfactory. Slight cases do best with an open air life and a moderate amount of exercise, as the appetite and general nutrition are thereby stimulated; but those who are severely anæmic often require to be kept in bed, and some only recover when this is strictly enjoined. A week or a fortnight is generally sufficient. The explanation of the beneficial effect of complete rest seems to be that the strain is taken off the heart and muscles, and consequently the digestion is improved, and food and iron are better absorbed.

Leucorrhœa, menorrhagia, or other source of blood loss must be carefully attended to. To prevent relapses, the appetite should be kept good and vigorous, and this is best attained by plenty of fresh air, and gentle, healthy exercise.

PERNICIOUS ANÆMIA.

This term denotes a condition characterised by intense diminution in the number of red blood corpuscles, and by the symptoms of severe chronic anæmia, usually terminating fatally.

History.—Cases of extreme fatal bloodlessness have been well and carefully described from time to time, as by Combe of Edinburgh (1822), who quotes a still earlier case (1684), by Channing in America (1843), and by numerous writers in France, Germany, and Switzerland. During the "forties" Addison made it familiar to the physicians and students of Guy's Hospital by his clinical teaching; and in 1855, in his monograph on "Disease of the Suprarenal Capsules," he gave a short but graphic account of its more apparent clinical features. Lebert (1868) named it "Essential Anæmia"; and later Biermer (1871) described fifteen cases of what he called "progressive pernicious anæmia," and this paper was the means of awakening new and widespread interest in the condition. Since then, numerous and valuable additions to our knowledge have been made by Pye-Smith, Hale

White, Bramwell, W. Hunter, Pepper, Quincke, Lépine, Hayem, and many other observers. H. Müller (1877) and Eichhorst (1878) published monographs which are still valuable as careful clinical and pathological records.

Etiology. — There is much difference of opinion as to whether pernicious anæmia is to be regarded as a disease in itself, "a morbid entity," or whether it is secondary to numerous other conditions. Addison describes it as "occurring without any discoverable cause; cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, and malignant disease," and Pye-Smith, Hale White, and others adhere to this definition, holding that anæmia, secondary to other causes, is not the true disease. Hunter also regards it as a "special disease both clinically and pathologically." On the other hand, Biermer, Quincke, Andrew, Müller, and many other clinicians, hold that it is a consequence of preceding conditions, some known, others unknown. Lépine believes that it follows always on causes predisposing to anæmia, but only in persons with weakness in blood-forming power; while Eichhorst thinks that in the majority of cases it is secondary, but in a few is primarily a blood disease. According to the present evidence before us, it seems to me most probable that it is always secondary, and that in those cases where it is apparently a primary lesion we have simply failed as yet to discover the preceding cause. It is now certain that the typical clinical picture which we associate with the name pernicious anæmia may occur in cases of atrophy of the mucous membrane of the stomach, in malignant disease especially of the stomach and bones, in tapeworm (*Bothriocephalus latus* especially), and in anchylostomiasis, where the parasite directly abstracts blood. Many cases also have been reported as occurring after prolonged diarrhœa, prolonged hæmorrhages, pregnancy, typhoid fever, malaria, syphilis, yellow fever, chlorosis, and great hardships. The majority of all such illnesses ordinarily run their course, however, without producing severe fatal anæmia, and we have no idea of what the superadded something is which determines the profound change in the blood. It has been surmised that in certain of these cases the bone marrow may have suffered permanent damage in its blood-forming functions, but postmortem the marrow usually shows signs of hypertrophy and increased activity. Some cases still remain apparently causeless, but it would be most proper to admit simply that we have not discovered the cause.

The defective condition of the blood must be due to deficient formation or increased consumption or loss of the red corpuscles, but even here unanimity of opinion is conspicuously absent. Bradbury, Pye-Smith, Mott, and others, hold that there is excessive and active destruction of the hæmocytes, Pepper that there is deficient manufacture, while Quincke and others are uncertain about it. Hunter has elaborated a theory that cadaveric bodies, formed in the intestinal canal, become absorbed and act as blood-destroying agents in the portal system. More recently he has expressed the opinion that the origin of such bodies is to be found in septic changes, often arising in connection with dental decay. I have formed the opinion that, in some cases at least, the external and internal hæmorrhages which are so common and extensive may lead to the excessive anæmia, once an anæmic condition has been established by ordinary causes. Grawitz proposes that the name pernicious anæmia should be retained only for those cases where, ante- or post-mortem, no sufficient cause can be made out for the extreme bloodlessness, while the others should be designated by such terms as "cancer with severe anæmia." The objection to this is that *intra vitam* both classes of cases often present exactly the same clinical symptoms; and even gross lesions, such as cancer of the bones, may only be revealed at the autopsy. At present it is quite impossible to give a dogmatic opinion on most of the points raised.

Morbid anatomy and pathology.—After death there remains the peculiar lemon-yellow tint of the skin, the fat everywhere has also a deep yellow colour, and the blood serum is slightly deeper in colour. There is marked fatty degeneration of the heart muscle (thrush-breast appearance), and marked pallor of all the internal organs and tissues. There may be only a few small hæmorrhages scattered here and there, but usually the number is very large, especially in the brain. They vary in size from a pin-head to a linseed, or larger, and there may be even large ecchymoses. Blood is frequently found under the dura mater. The bone marrow is altered, especially in the ends of the femur and long bones, the yellow marrow being converted into a deep red tissue, very rich in cells, many of them being large nucleated red cells. The liver, spleen, kidney, bone marrow, dura mater, brain substance, and fat, but especially the two first, show usually a very great excess of iron pigment, deposited in the form of an albuminous compound. It stains readily, black with ammonium sulphide, or blue with ferrocyanide of potassium and dilute hydrochloric acid. This excess of iron is generally thought to be derived from *active* destruction of the red blood corpuscles, and its presence is regarded as certain proof that such destruction does take place. I am of opinion, however, that the pigment can easily be accounted for by changes taking place in the blood extravasated into the tissues by the numerous internal hæmorrhages, as the same yellow pigment is also found in the organs and tissues after a hæmatoma or purpuric bleeding. Another source of the liver iron may possibly be found in the iron absorbed from the food and stored in the liver, but not used, as in health, to make hæmoglobin. In cases following external blood loss, or due to anchylostomiasis (if these can be reckoned as true pernicious anæmia), and in one case of unexplained origin, I have failed to find iron in excess, there being much less than normally. Cases which during life have had all the clinical symptoms of pernicious anæmia may reveal unsuspected gross lesions, such as cancer of the bones, atrophy of the stomach, etc., at the autopsy. Sclerotic changes in the spinal cord are sometimes found.

Symptoms.—It comes on slowly and insidiously; and as the patient insensibly gets accustomed to his bloodless condition, the anæmia is often very far advanced before any special feeling of malaise drives him to seek medical advice. On examination, the patient is usually found to be stoutish, with a good layer of subcutaneous fat, but with flabby muscles; the face and whole surface of the body are pale with a characteristic lemon-yellow tint, the sclerotics showing white and pearly by contrast. The mucous membranes have the same yellowish hue and are extremely pallid. The face or ankles may be puffy, from œdema. Extreme languor and debility with an apathetic temper are usually present, and there is excessive breathlessness on exertion. The heart is somewhat rapid and easily excited, while palpitation, hæmic murmurs, breathlessness, and the œdema, all denote the effect of the extreme anæmia on the circulatory system. The pulse is small, rapid, irritable, and of low tension. The dyspnœa is due partly to the want of blood and consequent lack of oxygen, and partly to the advanced fatty condition of the heart muscle.

The blood is pale and watery-looking, and it is often difficult to obtain a large drop by pricking the finger, this being due to the ill-filled condition of the vessels. It contains much more water than in health, and its total solids may be diminished to about half their usual amount, the loss falling chiefly on the red cells, and only slightly on the proteids of the serum. It coagulates badly, and the specific gravity is greatly lowered. Under the microscope the corpuscles are seen to form rouleaux somewhat imperfectly, to be very few in number, and to present a great variety of shapes and sizes. Some may be nearly twice the ordinary size (12 to 14 μ), others very small (3 to 4 μ), while the shape may be ovoid, cucumber-like, flask-like, comma-like, or otherwise distorted. In no other condition does poikilocytosis reach such a height as in pernicious anæmia. Nucleated red corpuscles can always be found. Dried films of the blood often do not stain well, or take on stains which normal corpuscles do not, thus showing some change in their protoplasm. Some observers regard these changes as a sign of increased activity in blood formation on the part of the bone marrow, while others look upon them as an evidence of debility. When a case first comes under observation, the hæmocytes usually number from about half to 1 million per cubic mm., but they may be much lower, and Quincke has recorded an instance in which they fell to 143,000. The hæmatoblasts are usually greatly diminished in number also. Some of the red corpuscles are distinctly pale, but on the whole the hæmoglobin is, according to my observations, diminished to an extent practically co-equal with the corpuscles. The latter are often said to contain an excess of hæmoglobin, but it must be remembered that the method of estimation is not one insuring extreme accuracy. The leucocytes are diminished in number, often very considerably, and especially the large multinucleated forms. It is stated by von Noorden that an increase of leucocytes precedes a change for the better in the number of red corpuscles.

Hæmorrhages from the nasal, intestinal, and other mucous membranes, under the skin, into the brain and retina, into serous membranes and all the viscera, are very common, and have a specially debilitating effect in cases already so severely anæmic.

Gastric dyspepsia, more or less severe, is always present; attacks of diarrhœa are very common and very troublesome and persistent at times. The patient's strength is so much reduced by them that they often precipitate the fatal issue. As regards the nervous system, the patients usually show a certain apathy and dull contentment. The minute hæmorrhages into the brain and spinal cord may cause slight motor and sensory disturbances, which pass off usually in a few days. Paresis or slight paralysis, or twitchings and passing disturbances of sight and hearing, are the most frequent of these symptoms. Subjective feelings of heat and cold, with rigidity, paralysis, ataxy, and other well-known symptoms of cord degeneration, have been met with during life, the cord changes having been verified by subsequent examination.

Feverish attacks are a constant symptom. The cause is unknown, but the temperature may rise, and for a week or two may oscillate between, say, 99° and 103° F., falling again to normal, or there may be a continuous slight elevation.

The urine is abundant, pale, and occasionally slightly albuminous, but sometimes becomes very highly coloured. This is due to excess of urobilin,

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which is a constituent of normal urine. Its presence denotes an excess of blood pigment in the circulation. The urea may be increased, diminished, or normal in amount. In some cases, no doubt, the increase depends on an increased metabolism of the nitrogenous tissues, which often occurs in anæmia, but the amount also depends on the nitrogenous food ingested, and is probably of no special significance.

In women there is amenorrhœa, and in both sexes distinct evidences of malnutrition are often shown by the condition of the hair and nails. Although the patients usually look well nourished, the excess of subcutaneous fat is accounted for by diminished oxidation and the absence of active nutritive changes.

Patients frequently improve under treatment, and may even appear to have quite recovered. Relapses are, however, extremely common, often occurring very soon, but they may be recovered from time after time. In this way the course of the disease may be very chronic, and a case may survive sometimes for years. Usually, however, death occurs in a few months after the patient is first seen, sometimes in a few weeks, and may come on quietly from extreme exhaustion and anæmia, or may be precipitated by diarrhœa or hæmorrhage. Pernicious anæmia is somewhat more common in women than in men, but if we except the cases occurring after childbirth and prolonged lactation, this would be reversed. It is most frequent in early middle age.

Diagnosis and prognosis.—The blood changes and the lemon tint of the skin, with the other clinical symptoms, make a diagnosis of severe anæmia easy enough. The great difficulty is to determine whether any known cause is present or not, and this, as may have been gathered, is always a matter of extreme difficulty. It would take up too much space to go in detail into all the possibilities, but the physician must keep these before his mind, and come to a decision by using his general medical knowledge. The prognosis is always grave. Temporary recovery is frequent, but relapse within a. comparatively short time is always to be feared. A very few cases seem to have recovered permanently; the statistics of recovery are somewhat unsatisfactory, however, owing to the fact of cases being lost sight of too soon.

Treatment.—As has been previously pointed out, cases of severe anæmia, due to cancer and other necessarily fatal causes, cannot always be separated clinically from cases which some physicians call "true" pernicious anæmia. Naturally, these fail to benefit under any system of treatment. Severe anæmia, due to intestinal parasites, is cured readily enough by anthelmintics. In all cases the greatest general care must be taken of the patient as regards diet, digestion, and exercise. Piles and any source of blood loss must be carefully attended to; and if diarrhœa should supervene, it must be treated without delay, and is always to be regarded as serious. The state of the gastric digestion often interferes greatly with a sufficient amount of food being taken, and the diet must therefore be as light and nourishing as possible. If able, the patient should be out in the open air in warm weather, but any active exercise must be avoided, as very slight exertion causes great breathlessness and fatigue, and strains the heart unduly. A warm hilly climate seems to agree best with most patients, if they are still in a condition likely to derive benefit.

As regards drugs, arsenic has come to be relied on as the most trustworthy means of stimulating the formation of new corpuscles. Under its use improvement very frequently takes place, and the patient often progresses until he may be said to have recovered. Unfortunately, in most cases, this improved condition is only temporary, and although a very few cases do remain well, a consideration of all the facts drives us to conclude that arsenic is not truly curative in its action, does not strike at the root of the disease, but simply improves the symptoms by causing an increase in the number of blood corpuscles. It should be given after meals, and may be cautiously pushed, if moderate doses do not take effect at first. Iron also seldom effects a permanent cure, although several observers have obtained temporary and even permanent benefit by its administration. Like arsenic, however, it is quite incapable of truly curing the condition; but its administration is advisable, on the grounds of supplying sufficient iron to meet the needs of the organism. Phosphorus has frequently been given, but with very little benefit.

Sandoz and others speak highly of the value of gastric and intestinal lavage, carried out systematically, and there is no doubt that in some cases very decided improvement results. In these the disease is supposed to have its origin in intestinal fermentation. On much the same principles Hunter and Gibson have advocated the administration of intestinal antiseptics, such as beta-naphthol, salicylate of bismuth, and salol, but in my experience the results of such treatment are usually disappointing. A strictly farinaceous diet has also been recommended by Hunter, with the view of reducing septic processes in the bowel. Improvement has also been reported under the use of red bone marrow, 1 or more ounces daily by the month. It is rather apt to derange digestion, and in my own experience no improvement has resulted from its use.

In all cases of pernicious anæmia we are accustomed to rely too exclusively on the condition of the blood as the sole criterion of the patient's improvement, and are apt to forget that this is probably only a symptom under which there is a deeper-lying cause, not touched by any of our remedies as yet. Even without treatment the blood condition sometimes varies a good deal, and hence it is often difficult to judge accurately the value of the remedies employed.

Transfusion of defibrinated human blood has very often been tried. In a very few cases remarkable benefit has resulted, but sufficient cases have now been reported, to enable us to form the opinion that it is generally without effect. Three to 6 oz., mixed with phosphate of sodium solution, are generally given by a vein; and as this amount can scarcely be supposed to efficiently supply the great deficit on the part of the patient, the theory has been promulgated that it may destroy or antagonise some toxine present in the blood of the recipient.

SCURVY.

Syn., Lat., *Scorbutus;* Fr. and Ger., *Scorbut.*

Scorbutus is a chronic morbid condition, affecting bodies of individuals, or occurring sporadically, the exact cause of which is unknown, but which follows on prolonged deprivation of fresh food, and is predisposed to and aggravated by hardships and bad hygienic conditions, characterised by moderate or extreme physical debility and mental depression, severe breathlessness on slight exertion, sponginess and ulceration of the gums with fœtid odour, large and small subcutaneous and internal hæmorrhages, and sero-sanguinolent effusions into serous cavities, ending fatally if untreated, but generally yielding readily to appropriate treatment.

Historical.—Immermann states that none of the ancient medical writers describe outbreaks of scurvy so precisely that the disease can be definitely identified, but nevertheless there is reason to think that its occurrence was not uncommon in armies and besieged towns. Pliny relates that an epidemic which attacked the army of Germanicus in the Netherlands was cured by the addition of a certain vegetable to the dietary of the soldiers, and this points almost certainly to scurvy. Later, there are accurate descriptions of outbreaks in European armies and besieged towns, in which the death-rate was very high, and which are directly traceable to the absence of fresh food, combined with severe hardships. In the fifteenth and sixteenth centuries it was widely spread and epidemic in Northern Europe, owing to the population living so largely on smoked and salted food without a proper supply of vegetables, but was much less common in the southern countries, where vegetable food forms a staple part of the dietary. Scurvy among sailors only came into prominence when the spirit of maritime enterprise led to long sea voyages being undertaken. When Vasco da Gama rounded the Cape in 1498, he lost from scurvy nearly two-thirds of his men; and when Jacques Cartier wintered in Canada, in 1535, practically every member of the expedition became more or less scorbutic, and many died. The disease must have been known to the Indians, for one of them advised the voyagers to drink a decoction of the bark and leaves of a certain tree, the beneficial result of which was immediate. The first recorded outbreak amongst English crews seems to have occurred in 1554, on a voyage to Guinea, but scurvy devastated our fleets and expeditions to a lamentable extent later on during the sixteenth, seventeenth, and eighteenth centuries. The prophylactic value of fresh vegetables, and especially of lemons, oranges, and scurvy-grass, was pretty generally known, but the voyages were very prolonged, and fresh fruit often very difficult to obtain, added to which the ships were greatly overcrowded, and cleanliness and ventilation little attended to. Nevertheless, Lancaster in 1601, on a voyage to the East Indies, kept his crew healthy by taking a supply of lemon juice with him; and Cook, in 1775, brought his ships home after a three years' voyage, without losing a single sailor from scurvy. But these and other experiences of the same kind were exceptional. Our early colonists in Virginia and New England also suffered severely from outbreaks of scurvy.

In the eighteenth century very exaggerated ideas prevailed among medical men as to the importance and widespread nature of scurvy, and many conditions in no way related to it were ascribed to a "scorbutic taint" in the blood. In 1753, James Lind published "A Treatise of the Scurvy," in which the whole subject is accurately and ably treated in all its aspects, and this was followed in 1762 by "An Essay on the most Effectual Means of Preserving the Health of Seamen in the Royal Navy." As a result chiefly of Lind's views, the naval authorities in 1797 instituted an improved dietary and the systematic use of lime juice, and these regulations soon practically banished scurvy from the British navy, the example being speedily followed by other nations, and more slowly by the mercantile marine. The substitution of steamers for sailing ships, and the consequent shortening of voyages, along with better food and accommodation, have had an enormous effect in diminishing the frequency of scurvy in our merchant service. Outbreaks were also at one time not uncommon in prisons, poorhouses, and other similar institutions, but at the present day scurvy, either at sea or on land, is a rare disease, although it still occurs under unusual conditions of dietary and hardship. It prevailed to a serious extent during the potato famine of 1848 in Ireland and Scotland, during the Crimean War, during the American Civil War, and during the siege of Paris in 1871, and is always liable to break out under similar conditions of faulty dietary, hardship, and unhygienic surroundings. It is sometimes seen in persons who have lived chiefly on tea and bread for a long time; and I have seen an outbreak among a gang of navvies who were working in an isolated district, and had been fed on smoked ham, cheese, and bread for some months.

Etiology.—Owing to its frequent occurrence in an epidemic form, scurvy was early thought to be an infective disease. Support is given to this view by the fact that outbreaks have occurred in ill-ventilated and unhygienic ships and buildings, while the inhabitants of consort-ships or neighbouring dwellings, living under exactly similar conditions otherwise, have escaped. Hence it is averred that the causation cannot be purely dietetic. But it may be urged as against this, that the unhealthy surroundings may simply assist and hasten the effects of a faulty dietary. Further, it is still in dispute whether contagion from person to person takes place, and, in spite of numerous attempts, no pathogenic organism has been detected. Nevertheless it is quite possible that future investigation may determine the immediate cause to be a specific germ. Meantime there is no doubt that the practical cause, so far as our knowledge goes, is to be found in a fault of the dietary, extending over a period of time. There has been much speculation regarding the particular element in the food which is absent, Garrod holding that it is the potassium salts, others that it is the vegetable acids, while it has even been maintained that the chief fault is merely a want of variety. Blame has been laid on the salt used in preserving food and on various articles of diet; but all these alleged causes may be dismissed as discredited theories. Scurvy has usually occurred in communities which have subsisted for some time on animal and vegetable food which has not been fresh. At sea this has almost invariably consisted of salted meats, dried legumes and dried cereals (peas, beans, flour, oatmeal, rice, etc.), the quality of which might be unexceptionable. Recovery from scurvy begins at once when fresh food, and especially fresh vegetables, are added to the diet. Further, children often become scorbutic when fed exclusively on preserved milk, while they remain perfectly healthy on fresh milk. Hence the conclusion may be drawn that fresh food, both animal and vegetable, contains some unknown substance essential to healthy nutrition, but which is gradually lost on keeping. It is known that the antiscorbutic properties of dried or preserved fruits or vegetables are not so marked as when they are completely fresh. It is probable, also, that salt meats and dried peas or cereals have not completely lost this hypothetical antiscorbutic constituent, for decomposed meat and mouldy biscuit are apt to cause scurvy still more rapidly. The cooking of flesh and of milk, and possibly also of vegetables, distinctly lessens their antiscorbutic value; hence fruits which may be eaten fresh and raw are most efficacious in treatment. This is probably also the reason why the prolonged deprivation of vegetable food may bring on scurvy in persons who are taking an abundance of fresh meat. Probably uncooked flesh is strongly antiscorbutic, but we have no exact knowledge or experience on this point, seeing that we have relied almost exclusively on fresh vegetables and fruits for treatment.

Scurvy is predisposed to by exposure to cold, damp, unhealthy surroundings, overwork and hardships; while cachectic conditions, such as chronic dysentery, malaria, alcoholism, and syphilis greatly weaken the resistance to it, as well as famine, military reverses, and depressing emotions generally. Children suckled by scorbutic mothers develop scurvy, and this points either to infection or to the absence of some necessary constituent of the milk.

Pathology and morbid anatomy.—The body may be well nourished or emaciated. Decomposition sets in very rapidly. The heart, the liver, and the other viscera are in a state of fatty degeneration. The mucous and serous membranes are marked by numerous ecchymoses and petechiæ, and the former may be eroded. No lesion of the walls of the blood vessels has ever been detected to account for the hæmorrhages. There is generally a lymphoid condition of the bone marrow. The muscles, the intermuscular spaces, the subperiosteal regions, the spongy bone, the subcutaneous tissue, the viscera, etc., may all be the seats of blood extravasation. If old, these are often hard from the formation of fibrous tissue. The spleen is large, soft, and flabby. The gums, the serous cavities, the bones, joints, etc., may be found in the conditions described under the clinical symptoms. The blood coagulates normally. An account of the postmortem examination of two persons who died of scurvy is given by Sutton in the *Clinical Society's Transactions*, 1871.

Symptoms.—A slight case of scurvy presents no very marked symptoms beyond some general feebleness, sponginess of the gums, one or more large subcutaneous ecchymoses, usually about the legs or lower part of the thighs, and petechiæ round some of the hair follicles, while recovery takes place very rapidly under treatment.

More severe cases have the following clinical history and appearances. The onset is more or less gradual, the patient becoming weak and listless, and complaining of pains in the muscles and elsewhere, of a rheumatic character; while the skin has a sallow tint, and is dry and furfuraceous, the lips and mucous membranes being bluish. The most typical features are connected with the gums and the subcutaneous hæmorrhages. The gums may be simply soft and spongy, bleeding on very slight injury, or they may be greatly swollen and ulcerated; or in severe cases they may be covered with fungating masses. The teeth may loosen, or even become detached; there is more or less pain on eating, increased salivation, and a fœtid or very horrible odour of the breath. The process begins round the crowns of the front teeth, where the gums become slightly swollen and red, and may be made to bleed on very slight pressure. Bleedings occur into its substance, ulcers form, these become swollen at the edges and fungating, and there may be formation of new fibrous tissue, which increases the swelling. At this stage chewing is impossible, and only liquid foods can be taken. In edentulous adults and in infants the gums are sometimes unaffected. The explanation of these effects is most probably to be found in the great tendency to bleeding on slight injury which is present in scurvy. The chewing of food causes slight pressure on the gums, especially at their junction with the teeth; small hæmorrhages take place into the tissues, which become swollen and painful, and diminished in vitality; the further sequence of events being attributable to infection from the numerous organisms present in the mouth, complicated by the general cachexia and hæmorrhagic tendency. The mucous membrane of the mouth is seldom ulcerated. Large subcutaneous hæmorrhages occur on the lower limbs and other parts exposed to knocks or slight injuries. These are hard, brawny, and tender; the skin over them presents every shade of colour, from purple to yellow, with which we are familiar in bruises, this being due to chemical alterations in the hæmoglobin. Large hæmorrhages also take place into the muscles and intermuscular spaces, under the periosteum of the tibia and other bones, into the eyelids and into the viscera. From the mucous tracts, especially of the nose and bowel, bleedings also occur, and from the lung and kidneys occasionally. Bleeding more rarely takes place into the meningeal spaces, the orbit, and other cavities; but the exact locality is probably dependent on traumatism more than anything else. These hæmorrhages, according to their site, may give rise to coma, hæmoptysis, melæna, hæmaturia, and so on. Small petechiæ of a dull red or purplish colour are present nearly always round the hair-follicles and sweat glands of the lower limbs, giving the skin a roughened feel; but they are also found all over the trunk and upper extremities in some cases.

There is more or less dyspnœa and palpitation on exertion; fainting occurs very readily, and even fatal syncope in bad cases. The heart is weak, and often presents hæmic murmurs. The appetite usually remains fair, but there may be anorexia or extreme voraciousness for food. Night blindness is an early symptom in some cases, and has been attributed to exhaustion of the retina being readily produced owing to excessive weakness.

Even in slight cases there is always a mild degree of anæmia, which may become severe in proportion to the loss of blood, until it is extreme. The number of white blood corpuscles is increased. But otherwise the changes are those attendant usually on anæmia. If an element of healthy blood be absent, its nature has not yet been determined. The urine is sometimes albuminous, and sometimes contains blood, but its known constituents remain practically unaltered.

If the patient remain under the conditions which have induced the disease, he passes into a state of extreme debility: the face assumes a haggard look, with sunken eyes; the spirits become depressed and gloomy: the legs become œdematous, and the cardiac weakness excessive. There are severe pains in the muscles, joints, and bones, which may result in complete helplessness. Blood-stained effusions, with inflammation of the serous membranes, are apt to occur in the pericardial, pleural, peritoneal, and joint cavities, and these may be accompanied with fever. Death takes place from asthenia, pneumonia, syncope, bloody diarrhœa, embolism, or some other complication, such as pericarditis, etc.

During severe scurvy the skin often ulcerates, especially over old scars or injuries; a brownish black slough forms, and there is an unhealthy looking discharge. Old fractures sometimes become ununited, apparently from absorption of the callus: the bones may necrose at parts from the pressure of subperiosteal hæmorrhages, or the cartilages may separate from the shafts. Occasionally tendons and joints become stiff, from fibrous adhesions having formed in blood clots effused round them, and this may give rise to lameness. The end usually comes only after some months of illness, but it may be hastened by hardships or the necessity for exertion.

Diagnosis.—There is no difficulty regarding diagnosis, as a rule. It is stated that beri-beri, peripheral neuritis, and purpura resemble scurvy in certain features; but the gums, the subcutaneous hæmorrhages, the history, and the rapid results of treatment, mark it off quite distinctly from every other disease.

Prognosis.—Slight cases improve materially in a few days, and even severe cases with no serious complications, cure in a few weeks under proper treatment. Dysentery, pericarditis, pneumonia, cardiac failure, and great general exhaustion are all serious as regards life.

Treatment.—Slight cases require nothing beyond good hygienic surroundings, rest in bed, and a proper dietary. The food should be easily chewed, simple and digestible, and should consist of fresh meat, fresh meat juices, fresh milk, and a large allowance of fruits and vegetables. Often all that is required is to make an addition of vegetable food to the ordinary diet. Improvement begins almost at once, and recovery may be complete in two or three weeks. Ulcerated gums may be touched with silver nitrate, and mouth-washes of potassium permanganate, alum, chlorine water, tincture of myrrh, etc., should be freely used. Gastro-intestinal disorders, pericarditis, ulcers, and all other complications, must be treated on general principles as they arise, but it cannot be too distinctly understood that all other measures are ineffective in the absence of fresh food. Astringents do not control the internal bleeding. The recovery of severe cases is often very tedious, and great care has to be taken, on account of the heart weakness and general debility.

Succulent fruits, such as oranges, lemons, and limes, just before they are quite ripe, have the greatest reputation as antiscorbutics, but apples, grapes, various kinds of berries, and probably all fresh acidulous fruits, are perfectly active. Potatoes, cabbage, greens, spinach, leeks, onions, celery, cress, lettuce, dandelion, scurvy-grass—in fact, most vegetables which are eaten raw as salads or cooked—are quite efficacious in preventing or curing scurvy. Expressed lime juice or lemon juice, preserved with about 10 per cent. of rum or brandy, is very largely employed, especially at sea, where fresh vegetable food is difficult to obtain, but it should never be used in preference to the latter. Its antiscorbutic qualities sometimes diminish greatly on keeping, and especially on exposure to the air, so that it should be stored in vessels of moderate size, which can be opened as they are required. Cider, spruce beer, and claret have all a reputation in preventing scurvy, but their influence is probably slight.

Milk loses its antiscorbutic properties completely or partially by being "preserved," peptonised, or thoroughly boiled. Sterilisation by heating is also said to be hurtful, but there is some doubt regarding this. Cooking probably also diminishes the antiscorbutic value of flesh and animal food generally. It is quite unknown on what components of fresh food its antiscorbutic properties depend, but dried and preserved foods of all kinds have this property to a much less degree, and gradually lose it entirely. Lind knew that "dried spinage prepared in the manner of hay" was of no value as a prophylactic. Scurvy can be prevented at sea by a full mixed diet. Fresh meat and vegetables should be obtained as often as possible, and in their absence preserved fruits and vegetables should be freely used. Apples, oranges, and lemons keep well and are easily carried. Good lime juice is quite efficient as a prophylactic, and by the terms of the Merchant Shipping Act each member of the crew receives one half-ounce daily, after being on salt provisions for ten days. In dieting bodies of men on land, care should be taken to ensure a sufficient supply of fresh food and especially vegetables, for whether the immediate cause of scurvy be a specific germ or a dietetic defect, it is quite certain that persons properly fed remain free from it under all circumstances.

INFANTILE SCURVY—SCORBUTIC RICKETS.

Scurvy sometimes occurs in young children who are being improperly fed. Attention has been especially drawn to its clinical features and comparative frequency by Cheadle and Barlow. It is usually met with in infants between 6 and 18 months old (much more rarely in older children), who are being fed on an exclusive diet of preserved milk, patent infant foods of one kind and another, or on milk which has undergone prolonged boiling. The child gradually becomes feeble and peevish, lies very quietly if left alone, but suffers severe pain on being handled. There is usually a good deal of gastro-intestinal disturbance, and the skin has an unhealthy earthy tint. The child lies with its legs and thighs flexed, afraid or unable to move them owing to the pain. Subperiosteal, intramuscular, and small subcutaneous hæmorrhages occur, with œdema and swellings round the joints. The legs are most commonly affected, but the region of the orbit and the ribs also suffer not infrequently. All these parts are extremely tender on being touched.

Hæmaturia may be present. The gums are as a rule little affected, the only indication sometimes being slight sponginess. The long bones or ribs may become detached from their cartilages, or necrosis may occur from pressure of the subperiosteal effused blood. It is often accompanied by a certain degree of rickets, but there is most probably no necessary connection between the two diseases. The prognosis is good, if proper treatment be carried out. The child should have plenty of fresh milk, fresh grape or orange juice, small quantities of mashed potatoes, and careful attention to its digestion and general health. Recovery usually has taken place in about three or four weeks.

PURPURA.

Purpura is a name applied to certain morbid conditions of unknown causation, in which the most striking symptom discernible is the occurrence of numerous small hæmorrhages into the skin. In the present state of our knowledge it is difficult to give a more exact definition.

Historical.—The term purpura has been used very loosely by medical writers, although the occurrence of purpuric bleeding has been noticed since very early times. Sauvages, in his "Nosologia Methodica" (1769), describes purpura senilis under the name ecchymoma melasma, and refers back to Galen. He also lays stress on purpura occurring in malignant fevers, and a mild form without fever, purpura maligna and purpura benigna of Schacher (1723). In the eighteenth century the term purpura alba was also applied, curiously enough, to sudamina and similar colourless eruptions, so that considerable confusion existed. In 1735, Werlhof of Hanover described purpura hæmorrhagica in full detail, and since his day the disease has been very generally recognised. Cusson (1727–1785) describes as purpura apyreta the occurrence of purple spots over the whole body, without itching, tumour, or other symptoms (purpura simplex). Graaf, in a thesis (1775), describes a case under the title, "De Petechiis sine Febre." Andrew Duncan also gives details of a case (1778), and proposes the name petechianosis for the condition; while Aikin calls it "hæmorrhage and purple spots without fever." In 1837, Schönlein published a description of peliosis rheumatica.

Etiology.—Cutaneous hæmorrhages, with or without bleedings from mucous membranes and into internal organs, occur in many well-known conditions, such as pyæmia, enteric fever, scarlet fever, measles, etc.; and in these they are attributed to the action of the toxines on the blood or small vessels. Snake poison, potassium iodide, phosphorus, and some other drugs may also cause purpuric bleedings, while in pernicious anæmia, leukæmia, tropical diarrhœa, scurvy, and numerous other diseases, larger or smaller hæmorrhages are not uncommon. In all these cases they constitute merely a symptom, usually, but by no means always, of subordinate importance, and it is quite possible and even probable that in purpura their significance is the same. In the latter, on the other hand, the bleedings constitute the most obvious symptom; and as we are quite ignorant of the underlying cause, it is convenient, as a provisional expedient, to include certain conditions under this heading. Different writers have classified them in various ways, but provisionally, for descriptive purposes, we may recognise three chief types, which have been named respectively purpura simplex, purpura hæmorrhagica, and purpura rheumatica. Most probably they will be proved to be specific fevers. A large number of micro-organisms have been described as occurring in purpura, but none of them can be regarded as proved to have any causative relation to the affection. The purpuric bleedings which occasionally occur in new-born infants (purpura neonatorum), in the aged (purpura senilis), and in convalescence from acute diseases, scarcely come under any of these headings, and are to be regarded as due to disease of the walls of the small vessels. Purpura is a rare condition, and generally occurs before the age of 35. It may occur in the perfectly healthy without warning, and is sporadic in its incidence.

Pathology and morbid anatomy.—Hæmorrhages, from the size of a small seed to that of a large or small coin, are found in the skin in all stages of the discoloration which extravasated blood passes through. In fatal cases, large or small extravasations are also found in all the viscera, and in serous and mucous membranes. They are due to rupture of small blood vessels. Altered blood pigment giving the chemical reactions for iron is also found more or less abundantly in all the viscera, and is derived from the extravasated red corpuscles. If the hæmorrhage has been severe, there may be marked anæmia. In some cases slight nephritis has been recorded.

Symptoms.—The disease is usually subdivided into several different clinical forms.

Purpura simplex.—The patient may be in perfect health, or may have been ailing slightly for some days, when a large number of small hæmorrhages suddenly appear in the skin of the legs and thighs, and sometimes in other parts. They vary much in number, and may be slightly raised, or level with the surface, but never disappear on pressure. This may be all; but successive crops may occur, and then we find the marks in all stages of discoloration. Any exertion, even rising from bed, may determine further bleedings. It is not known if there are corresponding internal hæmorrhages. Sometimes there is slight fever and a good deal of general debility. The prognosis is good as regards life, but sometimes successive eruptions go on in an exasperating way for many weeks. Purpura simplex may develop into the hæmorrhagic form, when the prognosis becomes more doubtful.

Purpura hæmorrhagica (morbus maculosus Werlhofii).—Hæmorrhagic purpura may begin with slight or with startling severity, the bleeding taking place into the skin, and into all the internal organs and tissues, as well as from the mucous membranes. Epistaxis especially is often very severe and uncontrollable, while bleedings from the palate and gums were regarded as very characteristic by the early describers of the disease. There may be no prodromal symptoms, or there may have been anorexia, lassitude, pain in the back, and slight debility for some days previously. Sometimes it may have begun with slight purpura. If the loss of blood goes on, it results in profound anæmia, with palpitation and dyspnœa on exertion. Hæmatemesis, melæna, hæmaturia, brain symptoms, eye symptoms, swellings of joints, etc., may all occur from bleeding into different parts. The temperature is usually elevated, sometimes to 105° F. even, but also there may be no fever.

Death may come on rapidly from hæmorrhage, pulmonary œdema, apoplexy, or exhaustion, but the hæmorrhages may cease, and recovery gradually take place. A severe hæmorrhage may occur without warning, and suddenly endanger life. Many cases are comparatively slight, the bleedings are not extensive, and recovery is complete in about a fortnight after they are first noticed.

Purpura rheumatica (peliosis rheumatica).—There are prodromal signs of general malaise, slight fever, poor appetite, and pains in the muscles and joints. The joints of the leg are especially liable to be affected, and synovitis is present with a good deal of swelling and effusion. The purpuric bleedings occur along with the joint attack, and may be confluent, so as to present the appearance of fairly large extravasations, but most of them are small. They are often a good deal raised, and the skin generally has sometimes the appearance as in a case of erythema exudativum, or erythema nodosum. There is often a good deal of subcutaneous œdema, especially in the feet, and the anæmia may be considerable. Occasionally there is endocarditis, sometimes albuminuria, and sometimes epistaxis. It is thought that this affection has a close connection with rheumatism, but it is probably a distinct disease, as salicylates make no impression on its course.

The prognosis is usually good, but relapses are very apt to occur. In some cases there is only one attack of the joint pains and hæmorrhages, but in others they recur at intervals, so that the illness may go on for months. It is very apt to recur in any person once affected.

Henoch's purpura.—Henoch has described a form of purpura which occurs usually but not invariably in children. The symptoms are, a purpuric eruption, swelling of the joints, blood in the fæces, vomiting, and colic, the attacks being apt to recur at intervals. Bleedings from other parts sometimes occur, with œdema and exudative erythema of the skin. The cause is unknown.

Diagnosis.—Purpura is distinguished from scurvy by the history of faulty diet in the latter, by the gum affection, and by the results of treatment; from hæmophilia, by the hereditary history chiefly, and a consideration of all the circumstances, but it is often difficult. In leukæmia, also, its diagnosis is sometimes difficult, as the earliest symptom may be a violent attack of purpuric bleeding, but the examination of the blood and the swollen glands usually lead to a correct diagnosis.

Treatment.—The patient should be strictly confined to bed, as merely getting up often seems to precipitate further hæmorrhage. The diet should be light, nourishing, and bland, and the bowels should be carefully regulated. Sulphuric acid and turpentine have both a considerable reputation for efficiency in stopping the bleeding, but their value is not accurately determined. Ergot, lead salts, calcium chloride, and iron persalts are also used, but without much benefit. Cold pressure, plugging, and the ordinary surgical means of controlling hæmorrhage may be employed where practicable. For prevention, great care should be taken to avoid overwork, exposure to cold and damp, strong excitement or emotional disturbances, and the general health and digestion should be kept at as high a standard as possible.

HÆMOPHILIA.

Syn., Hæmorrhagic Diathesis.

A congenital inherited tendency to severe spontaneous or traumatic bleedings, which lasts throughout life.

Historical.—Only a few scattered notices of the disease and of individual cases appear in the writings of medical authors, beginning with the Spanish-Arabian physician, Alsaharavi (12th century), until, in 1784, Fordyce described it as occurring in a group of persons who were blood relations. Shortly after, more detailed accounts of several "bleeder" families were published in America and elsewhere, so that Nasse in 1820 was able to give what is practically a full and modern description of the condition, and somewhat later the name hæmophilia was bestowed on it by Schönlein. Since then many interesting cases and some monographs have been published, that of Wickham Legg (1872) being the most important in the English language.

Etiology—Hæmophilia may arise *de novo* in a single member of a non-bleeder family from absolutely unknown causes, and this person may serve as a starting-point for its hereditary transmission. Otherwise, heredity is by far the most common cause, as its occurrence is largely confined to certain families and their offshoots, in some of which it has now been accurately traced for as many as four to seven generations. In a bleeder family the males are most liable to the disease, although some of them escape almost invariably. The most common form of inheritance is for the non-bleeder mother of a tainted stock to have bleeder sons, whilst the daughters escape personally, but in their turn transmit it to their sons. It may, however, be transmitted directly from father to son, from father to daughter, or from mother to both sons and daughters, or it may skip a whole generation, or may show other irregularities. The non-bleeder men of affected families who marry healthy wives usually beget healthy children, who do not transmit the tendency, but the non-bleeder women of the same families seem invariably to have bleeder offspring. Therefore the women of an affected family are the chief means of transmission, although they themselves may not suffer from the hæmorrhagic tendency. Males are affected in the proportion of about 92 per cent., and females 8 per cent., of all persons who suffer from hæmophilia in its typical and fully developed form. It is probable, however, that the proportion of women who exhibit a slightly exaggerated tendency to bleeding is much larger in these families than the latter figure would indicate.

Hæmophilia is undoubtedly on the increase, for although the actual bleeders tend to die young, and therefore to leave no offspring, yet its marked transmission through the non-bleeder women, who have no tendency to early death, is the important factor for its continuance. Added to this is the fact that such women show an average fecundity nearly double that of the ordinary population, and hence new centres for its propagation are continually arising.

The Anglo-Germanic races and Jews are said to be more liable to hæmophilia than the Latin and Asiatic peoples, but it seems to occur everywhere and in all classes, and our statistics are neither so wide nor so reliable that we can dogmatise on these points. As regards its essential cause we have no knowledge whatever.

Pathology and morbid anatomy.—In persons who die from the hæmorrhage intense anæmia and bleedings internally are the only post-mortem appearances. Careful examination of the blood vessels, blood, and viscera have in many cases given only negative results. In others, general hypoplasia of the vascular system has been described, the walls of the arteries have been found thin, and the endothelial cells of the capillaries swollen and proliferated. It is very doubtful, however, if these changes can be considered specific.

Regarding the blood, statements differ somewhat. At the beginning of a hæmorrhage it has been said to be normally coagulable; but recent observations seem to point to a slower and lessened coagulability. As the patient becomes more exsanguine the coagulability certainly becomes much less, and the blood very pale and watery. In a case under my care which proved fatal from hæmoptysis, the blood at first resembled what is seen in ordinary circumstances, but towards the end the fibrin separated out from it, as in whipped blood, and it showed not the slightest tendency to coagulate. In another case, where, however, there was almost continual chronic anæmia from hæmorrhage, I found that the red blood corpuscles broke down very readily under the action of reagents; but this is probably secondary to the anæmia. Whatever the abnormalities of the blood or vessels may be, we have as yet failed to discover them. Some of the joints usually show certain anatomical changes, in consequence of frequent bleedings into them. The synovial membrane and other structures may be stained by more or less altered blood pigment, there may be inflammation, and there may be alteration of the articular cartilages, even to erosion. Legg says that the ultimate effects resemble the changes seen in rheumatoid arthritis.

Symptoms.—The constitutional tendency to bleeding is almost invariably manifested during the first few years of life, sometimes apparently spontaneously, sometimes as the result of a trifling injury or operation. In a few cases the first hæmorrhage has been noticed much later (between ten and twenty years), but whether the tendency has here developed late or has been present all along, is difficult to decide. It is said that in some individuals it decreases after middle age. To outward appearance, and on medical examination, bleeders do not differ from the rest of mankind, nor have they any peculiar mental characteristics. The severity of the bleedings and the extreme liability to them vary much in the affected individuals. The hæmorrhages usually occur as the result of traumatism, although the injurious influence may be so slight as probably in many instances to pass unnoticed. The pressure of clothes, a wrench, a slight knock or jar, sudden exertion, and strain, may all probably determine bleedings into the skin, joints, and viscera; or, externally, cuts, abrasions, blows, slight operations, circumcision, drawing a tooth, opening an abscess, leeching, blistering, not to mention more serious lesions, have all given rise to excessive and uncontrollable hæmorrhage. Large operations are deadly. Vaccination, curiously, is stated to be practically safe.

The spontaneous bleedings are stated to be preceded for some days by feelings of fulness and throbbing in the head, giddiness, and flushings—the symptoms of plethora, in short. Epistaxis is by far the most common; but they also occur from the gums, stomach, bowel, urogenital organs, lungs, kidneys, and even rarely from the skin. Subcutaneous and interstitial hæmorrhages are also common, and may form large hæmatomata. Menorrhagia is uncommon, except at puberty and at the menopause, while parturition is only very exceptionally a source of danger. In all these the amount of bleeding varies very much, the blood coming fast and abundantly in some cases, and slowly and in small quantity in others, but very persistently in all. The bleeding is oozing and capillary in character, and if pressure be applied, the blood wells up at the sides of the finger or tampon, or penetrates into the surrounding tissues. As it progresses, either rapidly or slowly, the patient's skin becomes extremely pallid, with the purple veins showing through prominently, and there is much prostration. Death may occur suddenly and quietly from syncope and loss of blood; or the bleeding may cease, when, after a period of extreme anæmia, with a strong tendency to sleep, the patient begins to make blood, and gradually recovers. It is said that hæmophilics tolerate loss of blood better, and make it good more quickly, than ordinary persons; but in two cases which I have observed carefully I cannot say that I found this to be the case, as they were both chronically anæmic.

Persons who suffer from typical hæmophilia are very subject to hæmorrhages into some of the joints, and this is so troublesome that it constitutes a special feature of the disease. The bleedings probably depend on slight traumatism, or may be spontaneous perhaps, and they give rise to symptoms, varying from slight pain and stiffness up to severe pain and swelling, with bloody fluid in the joints. The knee is most commonly affected, but the ankle, elbow, hip, and shoulder-joints also suffer frequently. The pain and swelling render the joint temporarily stiff and useless, while repeated attacks lead to permanent changes.

Apart from the extreme and typical form of hæmophilia, some members of bleeder families seem subject to hæmorrhage without its ever being so severe as to cause anxiety as regards life. For three years I once had under observation a servant girl, who about every three months had a severe attack of menorrhagia, or an epistaxis, or hæmatemesis. The bleedings always came on suddenly and profusely, and usually stopped without any very special treatment. Her maternal grandfather had been subject to severe bleedings, her mother had once nearly died from hæmorrhage after extraction of a tooth, and her only brother was also a bleeder. She knew nothing more of the medical history of her family, but was well aware of the tendency of its members to bleed.

Diagnosis.—The family history is all-important. If the mother belongs to a bleeder family, petechial and other hæmorrhages occurring during the first three years of life almost certainly stamp the child as hæmophilic. Their occurrence after the most trifling abrasions or injuries, and the difficulty in stopping them, are very characteristic. Diagnosis is not so easy in modified cases, especially in women, and in cases occurring in non-bleeder families. It is only after repeated hæmorrhages that a conclusion can be come to, as a single severe hæmorrhage might quite well be an accidental circumstance. The joint affections give considerable aid to arriving at a decision in some cases.

Prognosis.—The majority of severe cases die in childhood before the seventh year. Some of them, however, survive to middle life, and even to old age, with their hæmorrhagic tendency still unabated. The outlook for such is very gloomy; but it no doubt depends largely on the kind of life they are able to lead, and on accidental circumstances, both of which may favour them. Most of them die sooner or later of hæmorrhage. Slight cases seem to lose the tendency after middle life. In boys the prognosis is worse than in girls.

Treatment.—This disease could be largely stamped out if the women of bleeder families refrained from having offspring, as also the men in those families where it is transmitted through the males as well as through the females. Children who are the subjects of hæmophilia must be carefully guarded from injury, all rough play and sports forbidden, and as they get older a quiet indoor occupation selected for them. The greatest precautions should be taken to avoid wounding or cutting.

When bleeding does occur, it must be treated on general surgical and medical principles, by compression, cold, styptics, and ergot. Calcium chloride (15 grs. three or four times daily in flavoured water) has been strongly recommended, and lately the subcutaneous injection of gelatin solution. The patient should be kept quietly in bed on a very bland diet, and saline cathartics should be given very freely. The joint conditions, when they occur, must also be treated by rest and on general principles. Morphine or ergot can be administered, if necessary, by hypodermic injection, without risk of bleeding.

RALPH STOCKMAN.

DISEASES OF THE BLOOD AND GLANDS.

LEUKÆMIA.

Syn., Leucocythæmia.

THIS is a chronic disease, the cause of which is not known, characterised by hypertrophy of lymphoid tissue all over the body, and by a great increase in number of the leucocytes of the blood, with diminution of the hæmocytes, and tending to end fatally.

History.—In 1845, Hughes Bennett described two cases with enlarged spleen as "suppuration of the blood," and later in the same year Virchow independently published an accurate account of the condition, rightly attributing the peculiar appearance of the blood to a large increase in the number of leucocytes. In papers immediately following he greatly extended his observations, and drew attention to several cases of "white blood" which had been described earlier. He called it leukæmia, while Bennett later gave it the name leucocythæmia, both derived from its most prominent symptom. Virchow recognised the implication of the spleen and lymphatic glands, and Neumann (1868) showed that the bone marrow is also involved. Mosler (1872) gave an accurate description of the different kinds of white cells present in the blood. Besides these authors, many others have made valuable additions to our knowledge, for although the disease cannot be said to be a common one, it has aroused a most unusual share of interest and attention in all countries.

Etiology.—In the absence of definite knowledge regarding the cause of the disease, exposure to cold, malaria, syphilis, injury, and other factors have all been invoked, but these are so common, while leukæmia is so comparatively rare, that they cannot be regarded as satisfactory explanations of its occurrence. It has been surmised that it may be infective in character, although no confirmatory proof has been given in this direction, but rather the contrary. It has not been possible to infect animals from man, by direct inoculation of the blood or diseased organs; and although many domestic animals are subject to the disease, attempts to transplant it to others of the same species have hitherto proved futile.

Pathology and morbid anatomy.—The condition of the blood will be described with the symptoms. Post-mortem, the clots are numerous, and in the heart and large veins often have a pinkish or a greenish pus-like appearance, due to the large number of white corpuscles and the peculiarities of coagulation. Hæmorrhages into serous and mucous membranes are usually present. The spleen is much enlarged, weighing very commonly 4 to 6 lb., and Bristowe says it may weigh as much as 20 lb. In the early stages, there seems to be simply a hyperplasia of all its elements, but later it becomes hard and fibrous in consistence, from increase in the trabeculæ, and the Malpighian bodies are then compressed till they disappear. The capsule is much thickened, and shows evidences of perisplenitis. On section it shows a deep, dull red colour, with hæmorrhages in all stages, and whitish lymphoid tumours scattered through it. It contains, in all the cases I have examined, a large amount of iron pigment, which stains readily with ammonium sulphide, or with ferrocyanide potassium and hydrochloric acid.

The lymphatic glands are moderately enlarged, and may be quite soft, or hard and fibrous from increase of their connective tissue. There are no abnormal changes in structure. The bone marrow, both red and yellow, is replaced by pink, greyish, or peculiar pus-like greenish masses of white cells. The bone may be thinned, and even swollen and tender at points. The change seems to consist in the great increase of the colourless marrow cells; lymphocytes, granular cells, and myelocytes being all present along with ordinary red and nucleated red cells.

The lymphoid tissues of the tonsils and mouth and of the intestine may be greatly increased, and lymphoid growths may be found in almost any part of the body. Leucocythæmic tumours are rare, however, and when present arise from aggregation of leucocytes, which lie in a delicate network and multiply by fission. The liver enlargement is usually due to diffuse leukæmic infiltration, lying between the cells and along the vessels, but sometimes there are small tumour-like patches. Everywhere the capillaries may be crowded with leucocytes. The liver shows a large quantity of iron pigment.

Symptoms.—The disease takes its origin in the spleen, lymphatic glands, and bone marrow chiefly; and although it is customary to speak of a splenic, lymphatic, and myelogenous form, according to the organ most severely affected, yet it never happens that any one of these is affected to the complete exclusion of the others. At the same time the lymphoid tissues of the tonsil and pharynx, and of the solitary and agminated glands of the intestine, are affected, and in fact practically all the lymphoid tissue in the body. It may occur at any age, but is much commoner in men and in early middle life than in women, or at other ages, although it is met with in children and rarely in advanced life. Its onset is very gradual, and attention may be first drawn to it in many ways. Sometimes the enlarged spleen or lymphatic glands are the earliest symptoms to attract attention, but the anæmia, or the increasing weakness, or a hæmorrhage, or some other of the numerous symptoms, may cause the patient to seek medical advice. As the disease is generally well developed by this time, suspicion of the state of affairs is usually aroused, and a microscopic examination of the blood leads at once to a positive diagnosis. On pricking the finger, the drop of blood obtained is turbid and pale, and coagulates badly; in extreme cases it may look like sanious pus, and on placing it under the microscope the number of white blood corpuscles is seen at a glance to be enormously increased. In proportion to the red they may be one to twenty, but are found in all proportions even up to equal numbers, and may even exceed the hæmocytes (Robin). A notable increase of leucocytes should always cause suspicion, as cases of leukæmia have been recorded where the proportion was one to fifty. The red corpuscles as a rule are not excessively diminished in number, and, in the early stages at least, usually amount to between 2 and 3 millions per cubic mm., but later they may fall to under 1 million. The hæmoglobin is proportionately diminished. The hæmocytes form good rouleaux, but many small and somewhat deformed corpuscles are present, while nucleated red cells of medium and large size (megalocytes) are common. The condition of the red cells is probably a consequence of the marrow changes. As regards the white cells, all the usual forms are present in varying proportions; and in addition one also finds large mononuclear forms with fine granular masses of protoplasm, very large and very small eosinophile cells, and the distinctive large marrow cells (myelocytes). According to the preponderance of the different kinds of white cells, it has been supposed possible to say definitely whether the lymphatic glands, spleen, or bone marrow is the organ most seriously implicated, but extended observations have shown that no reliance can be placed on this. The large white cells show very sluggish amœboid movements, and often contain colouring matter, which is either hæmoglobin or some nearly allied compound. Charcot's crystals (spermin phosphate) sometimes develop in the blood after its withdrawal. Ehrlich has stated that in leukæmia the eosinophile cells become very numerous, whereas this is not the case in mere leucocytosis, but such cells are common in many forms of severe anæmia, and are therefore not of exact value for differential diagnosis. Albumoses are found in excess in the serum, and account for its slow coagulation; while leucin, xanthin, and other bodies, which seem to be derived from breaking down of the leucocytes, are also in excess. If a moderate quantity of blood be withdrawn from a vein, and allowed to coagulate in a narrow glass vessel, it is seen, as soon as the serum separates out, that the fibrin and white corpuscles form a whitish mass on the upper surface of the red clot, thus giving the coagulum a very distinctive appearance. The specific gravity of the blood may be greatly diminished, depending chiefly on the degree of anæmia.

The enlarged spleen causes a feeling of weight and dragging in the left side, sometimes accompanied by dull pain, sometimes painless, and friction may be felt or heard over it. On inspection, the abdominal wall is seen to be bulged forward by the enlarged viscus, which extends down and across the abdomen, sometimes even into the pelvis. It feels hard, has a quite distinct outline, and may vary in size from time to time, probably according to the amount of blood it contains. Painful attacks of perisplenitis may occur from time to time. The lymphatic glands all over the body may be enlarged, either slightly or considerably, but they remain separate and are not matted together. They are usually a little tender on pressure, but this varies. The implication of the marrow often gives rise to no outward symptoms, but sometimes to tenderness on percussion, especially in the case of the sternum and flat bones.

Hæmorrhages are very common, and sometimes so universal and extensive as to rapidly cause death. They are often a very early symptom, and may occur from any mucous surface, or into any part of the body, but epistaxis is by far the most frequent.

The heart and circulation show the usual symptoms consequent on chronic anæmia. The former is excitable and rather rapid, hæmic murmurs develop, and more or less œdema. It may be pushed up by the enlarged spleen. Dyspnœa is often complained of, arising partly from the blood condition and partly from pressure on the thoracic organs. The nervous system generally is not specially affected in any way, except as a result of the anæmia, but vision and hearing are sometimes interfered with by small hæmorrhages into the eye and ear, retinitis occurring sometimes as a consequence of the former. I have also seen paralysis of the facial nerve, from pressure due to a hæmorrhage. Excessive sweating may be present. Gastric digestion is never really good, and very considerable dyspepsia is often complained of. Diarrhœa is very common, and may greatly depress the general strength of the patient. It is said to be due to lymphoid growths in the bowel, but this has been denied, as it does not necessarily occur in cases where such growths have been abundant. There may be hæmatemesis or melæna, and sometimes great discomfort after eating, or severe constipation, from the pressure of the enlarged spleen on the stomach and bowels respectively. The liver is at first normal in size, but later it usually becomes very greatly enlarged. There may be jaundice, either from catarrh of the bile ducts or from pressure exercised by enlarged glands, and ascites from similar pressure on the abdominal veins may be present. Fever may be absent, but as a rule there occur irregular febrile attacks, or the temperature may show a hectic character throughout the illness (101° to 103° F.).

The urine is not necessarily much altered, its condition depending greatly on the general condition of the patient. There is always a great excess of uric acid, derived from the nucleo-albumin of the leucocytes. Amenorrhœa or irregular and scanty menstruation is the rule in women. Priapism, said to be due to thrombi forming in the veins of the penis, is sometimes troublesome. Death may occur from asthenia, or from hæmorrhage, or from persistent diarrhœa; while apoplexy, pneumonia, pericardial effusion, rupture of spleen, or other intercurrent complication, may precipitate the fatal issue.

Diagnosis.—The examination of the blood, taken along with the symptoms and general condition, should enable a positive diagnosis to be made. There is no difficulty in advanced cases, in others it has to be distinguished from pseudo-leukæmia, splenic anæmia, malaria, and leucocytosis.

Prognosis.—This is always bad. It is possible that very early cases may have recovered, but cases where there is no doubt about the diagnosis seem always to end fatally. It is sometimes very chronic, and temporary improvement, with or without treatment, often occurs. Rapid increase of the white corpuscles, or decrease of the red, diarrhœa, and fever, are bad symptoms. Death may occur at any time from intercurrent causes, but one or two years is the usual duration.

Treatment.—General measures, such as careful dieting with nourishing food and alcohol, rest from active work, and good climatic conditions, tend to husband the strength. Dyspepsia, diarrhœa, anæmia, and other accompaniments must be treated on the usual lines.

To directly affect the disease, arsenic has been largely given, and in a certain number of cases it effects an improvement by diminishing the white and increasing the number of red cells. It should be cautiously pushed, until large doses are being taken. Its direct injection into the spleen does not offer any special advantages. Quinine has also been largely given, but with doubtful benefit. Bone marrow, spleen, lymphatic glands, and extracts made from them, have now been pretty extensively used, but I have never seen the slightest improvement take place under their use. Counter-irritation over the spleen, with biniodide of mercury ointment, or iodine, does not cause any visible improvement. Splenectomy hitherto has always been fatal in true leukæmia. During attacks of perisplenitis, opium and hot fomentations relieve the local pain.

PSEUDO-LEUKÆMIA.

Syn., Hodgkin's Disease; Lymphadenoma; Anæmia Lymphatica; Malignant Lymphoma.

This affection is a chronic condition of unknown causation, in which there is enlargement of the lymphatic glands and of the spleen, with secondary lymphatic growths in other parts, a decrease in the number of hæmocytes, and no marked increase of leucocytes, and tending to end fatally.

History.—Many cases of this disease had been previously described as scrofula, and as fatal enlargement of the lymphatic glands, but in 1832 Hodgkin gave an account of it as a distinct morbid condition. Since then, Wilks, Gowers, Virchow, Cohnheim, Trousseau, and many others have described cases, or discussed the disease in general terms, and have bestowed on it one or other of its numerous synonyms.

Etiology.—Great differences of opinion prevail as to the causes and pathological relationships of pseudo-leukæmia, and it is certain that no definite conclusions can now be arrived at until our investigations and observations have been greatly extended. Syphilis, tubercle, throat affections, and other causes of local or general glandular enlargement have been blamed as the starting-point, as well as cold, over-exertion, and the other common conditions which predispose to disease. To this the answer is at once ready—that all these are extremely common, while lymphadenoma is rare. It has been thought by some to be very closely related to leukæmia, and in a few cases has apparently developed into it; but in these there may be doubts about the first diagnosis, and certainly many cases run a very chronic course, without ever becoming true leukæmia. It has also been supposed to be a form of tuberculosis, and tubercle bacilli have certainly been found in the lymphatic glands, but there is always the probability that they are something superadded and accidental to the real disease. From the analogy of the symptoms and course of the disease, it has also been conjectured to be bacterial in origin, and this is a very probable hypothesis, although no positive evidence in its favour is forthcoming, for the present at least.

Morbid anatomy and pathology.—The lymphatic glands are soft and simply enlarged, or they may be hard and fibrous. In the former cases the lymphocytes are numerous, in the latter not so. The spleen is always moderately enlarged, and here there may be a simple hyperplasia, or whitish lymphoid tumours may occupy its substance, and greatly alter its shape if they be large. Numerous old hæmorrhages are generally scattered through it. Small adenoid tumours or adenoid patches may be found in the liver, in the whole of the alimentary canal, in the bone marrow, lungs, kidneys, suprarenal capsules, thymus, etc., while the tonsils may be greatly enlarged. Very seldom these tumours ulcerate in the bowel, or they may caseate.

Symptoms.—The initial symptoms may be a general loss of strength and some anæmia, but as a rule the first thing to attract attention is a painless enlargement of the lymphatic glands, usually on one side of the neck. The axillary glands, or rarely the inguinal, may be the first to enlarge. At the beginning each gland is quite distinct, and the skin is freely movable over them; but as the disease progresses they become much larger, and get matted together and adherent to the skin from periadenitis, or from extension of the tumour growth. They then form irregular masses as large as the fist, or even larger, and cause considerable deformity. The bronchial, mediastinal, and all the other lymphatic glands may gradually become involved, and may form very considerable tumours. These may give rise to the most diverse pressure symptoms, according as they affect the vessels and nerves of different parts, the œsophagus, trachea, heart, lungs, etc. Addison's disease may be induced when the suprarenal capsules are pressed upon or otherwise involved. The enlargement of the glands continues painless, unless they begin to grow very rapidly, when a certain amount of tenderness may be induced. They usually do not enlarge simultaneously or equally, as certain groups may grow at quite a disproportionate rate compared to others. Lymphatic tumours may also develop in the spleen, liver, lungs, skin, etc., and may give rise to numerous and varied symptoms. The spleen is usually somewhat enlarged, and may extend well beyond the costal margin, but seldom further. Lymphadenomatous growths in it may alter its shape.

The red blood corpuscles are always diminished in number, but in varying degree, and not necessarily very much at first. Microcytes and poikilocytes are present in fair numbers, but the changes in size and shape are not a prominent feature. The hæmoglobin is diminished proportionately to the corpuscles, and the specific gravity is also lessened. Any immediate cause for the anæmia is quite unknown. It gives rise to fatty degeneration of the heart, languor, œdema, amenorrhœa, and all the familiar circulatory symptoms. The blood coagulates badly, and hæmorrhages are common from the nose, as well as from or into any other part, but they are never nearly so marked a feature as in leukæmia. As a rule the leucocytes are not increased in number; all the ordinary forms are present, and there may be a slight excess of lymphocytes in some cases. An irregular febrile temperature is very often present, as in all severely anæmic conditions;

it may be remittent or intermittent, and is often a very early symptom. Digestion is always poor, and lymphoid growths in the walls of the alimentary canal and in the glands may lead to severe indigestion, diarrhœa, jaundice, and other complications. The liver may preserve its usual size and shape, or may be slightly enlarged. The urine presents nothing unusual as a rule.

The duration of Hodgkin's disease may be extremely chronic, a few glands continuing enlarged, sometimes for years, before others become involved. Five years seems to be about the extreme limit (Gowers) in the one direction, and five or six weeks in the other, but about two years is a very usual time for the disease to last previous to death. It is most common in young adults, and more so in men than in women. Death occurs from general exhaustion, and towards the end the anæmia may become very intense. Pressure of the tumours on vital organs, diarrhœa, acute pneumonia, pleurisy, or other complications, may carry off the patient.

Diagnosis and prognosis.—The diagnosis is often very difficult, especially if the mediastinal or other deep-seated glands be first involved. Scrofula, leukæmia, and syphilis are the most likely diseases for which it may be mistaken, but a differential diagnosis from these is usually attained without much difficulty. If the disease is acute or well marked, the prognosis is very bad. Marked fever and marked anæmia are always unfavourable signs. Some cases seem to recover spontaneously, or under the remedies employed.

Treatment.—The general dietetic and hygienic treatment is the same as in leukæmia, nor does the specific treatment differ much. Here also arsenic is the only drug on which one can place any reliance. Phosphorus, iodine, and iodides do not effect much improvement on the condition, nor do bone marrow, spleen, lymph glands, thymus and thyroid glands, or preparations made from them. If the enlargement be confined to a few glands which can be removed, they should be extirpated early, and without any delay. If the enlarged glands are more generalised, their removal is impossible.

SPLENIC ANÆMIA.

A progressive anæmia with enlargement of spleen and liver, accompanied by irregular febrile attacks and hæmorrhages, often very chronic and ending fatally. It is very doubtful, however, if this is a distinct disease, or only a purely splenic form of pseudo-leukæmia. Most observers incline to the latter view.

History.—Wilks (1861) described a case of this kind, and had previously referred shortly to the condition in the text-book of pathology written by himself and Moxon. Cases have also been described by Wells, Squire, and others, but little attention was excited until the appearance of Banti's monograph, in which he recorded three cases of his own, and made a summary of previous observations. In 1891, Bruhl published a long paper on the subject; in 1896 it was discussed at the London Medical and Chirurgical Society, and in the same year at the meeting of the British Medical Association. In France it has been called primary splenomegaly; Wilks described it as simple hypertrophy of the spleen, while the terms splenic pseudo-leukæmia, splenic cachexia, idiopathic splenic tumour have also been employed.

Etiology.—The cause is not known. It has been attributed to injury of the spleen, to syphilis, and to rickets, but I have seen cases in which all such constitutional diseases could with certainty be excluded. It is commonest in early adult life, and especially in the male sex, but it is met with at all ages.

Pathology and morbid anatomy.—The spleen has been found to weigh from about 2 to 13 lb. There is great increase in the fibrous trabeculæ, with atrophy of the Malpighian bodies. It contains numerous infarcts, and the sinuses are engorged with blood. If the cirrhosis be very intense, the blood sinuses may be almost obliterated. There is usually an increase also in the fibrous tissue of the liver, and the bone marrow has been found slightly hypertrophied. Slight hyperplasia of the lymphatic glands has also been reported. As regards free iron pigment in the liver and spleen, it seems to be sometimes absent and sometimes present, but never in excessive amount as in pernicious anæmia.

Symptoms.—The onset is very insidious, with gradual loss of strength, enlargement of the spleen, and anæmia. There is difference of opinion as to whether the anæmia precedes or follows the splenic enlargement, but by the time the patient comes under observation both are usually well pronounced. Epistaxis, or bleeding elsewhere, is a common early symptom. The spleen becomes greatly enlarged and painful at times, and there may be sharp attacks of perisplenitis with severe pain and fever. Apart from these, however, the temperature inclines to be feverish. The blood shows no specific changes, beyond a diminution in the number of red corpuscles and a proportional diminution in the hæmoglobin, along with slight poikilocytosis. The hæmatoblasts and white corpuscles seem normal. Hæmorrhages are common from the nose, stomach, bowel, and gums, and also into the retina and the viscera. According to the amount of anæmia, the patient may suffer severely from general weakness and the usual circulatory symptoms, or may feel fairly comfortable. There may be severe dyspepsia and diarrhœa, the liver is moderately or considerably enlarged, and attacks of jaundice are not infrequent, and are unaccompanied by any diminution in the number of red blood corpuscles. The patient may look fairly well, or severely cachetic, according to the general condition. Death occurs from asthenia, or from hæmorrhage, diarrhœa, or other complication.

Diagnosis and prognosis.—The diagnosis may be regarded as difficult or easy, according to the view taken of its nature. The clinical symptoms are definite enough, but we have no clear conception of its relationships to Hodgkin's disease, leukæmia, chlorosis, and other anæmic conditions. It is always fatal. The duration of recorded cases has been from three months to six years, but about two years is an average time.

Treatment.—Treatment is not very successful, but is to be conducted on the same lines as in Hodgkin's disease and in leukæmia.

ANÆMIA INFANTUM PSEUDO-LEUKÆMICA.

This condition was described by von Jaksch (1889), and is probably a congeries of clinical symptoms following on rickets, syphilis, diarrhœa, etc., rather than a distinct disease. There is considerable enlargement of the spleen, with severe anæmia and marked leucocytosis. The white cells may be to the red as one to twenty, or one to twelve, but the prognosis is fairly good notwithstanding. Under general tonic treatment with cod-liver oil, iodide of iron, quinine, and so forth, the spleen gradually diminishes in size, and the blood improves. If death occur, there is no evidence of leukæmia in the organs, but simply hyperplasia of the spleen, some enlargement of the liver, and the bone marrow rather more red than is normally the case.

RALPH STOCKMAN.

THE THYROID GLAND.

MYXŒDEMA.

This term comprises a series of symptoms following on partial or complete absence of the thyroid secretion, in consequence of atrophy or removal of the gland.

History.—Although isolated examples of this disease had been previously observed, we really owe its recognition to the late Sir William Gull, who in 1873 described five cases as "A Cretinoid State supervening in Adult Life in Women," and gave a very full account of the chief clinical symptoms. In 1878, William Ord. published a description of two cases, greatly extending our knowledge of the symptomatology and pathology. In one of these cases he performed a post-mortem examination, and found that the thyroid gland was in a state of fibrous degeneration; he also described a subcutaneous deposit of myxomatous tissue, and, in reference to this, gave the disease the name by which it is now universally known. In consequence of these descriptions, cases were immediately recognised in all civilised countries, and the disease became widely known. In 1883, Reverdin of Geneva observed the same symptoms after removal of the thyroid gland, and named the condition "post-operative" myxœdema, while Kocher described it as "cachexia strumipriva." The results following on total removal of the thyroid gland, both in man and animals, had, however, been known long previously, but had never attracted general attention. In 1887 a committee of the London Clinical Society reported that myxœdema, post-operative myxœdema, and cretinism, are all due to disease or removal of the thyroid gland, with consequent loss of its secretion. In 1891, Vassale found that the subcutaneous or intravenous administration of thyroid secretion to animals from which the gland had been removed, prevented the cachexia usually following on such removal. Previously to this, various surgeons had attempted to transplant the sheep's thyroid gland to human beings suffering from myxœdema, but it always atrophied, and any improvement had been very short-lived. In 1891, Murray of Newcastle showed a case which had been cured by the subcutaneous injection of a glycerin extract of sheep's thyroid, and in the following year Howitz, and later Mackenzie and Fox, intimated almost simultaneously that the raw or cooked gland given by the mouth was equally efficacious. Efforts were at once made to prepare the gland in more compact and palatable form, and at the present time the dried and powdered thyroid of the sheep, deprived of fat, is almost universally employed, although liquid preparations are also used, and preparations of the so-called active principle.

Etiology.—As will have been gathered from the foregoing historical account, the disease is due to partial or complete absence of the secretion of the thyroid gland, in consequence of atrophy, disease, or extirpation. In adults this gives rise to myxœdema, in children to cretinism. It is possible that in some cases the distinctive characters of the secretion have suffered a change. The exciting causes are obscure in a large proportion of cases, but the secreting structure of the gland seems to degenerate after many acute infectious diseases, after inflammation, and after cold and exposure. The prolonged use of iodine preparations seems occasionally to have induced atrophy; and syphilis, mental shock or depression, frequent pregnancies, goitre and exophthalmic goitre, and tumours in the gland, have all preceded myxœdema, and have apparently had a direct etiological connection with it in these cases. It has also been observed to occur with unusual frequency in certain families. It occurs much more frequently in women than in men, the proportion being generally given as six to one, but it is probably higher. The reason for this preponderance has been sought in the unexplained connection between the generative organs in the female sex and the thyroid gland, which often enlarges during pregnancy and menstruation, and on the occurrence of uterine tumours. The disease is most common in middle life (30 to 50), but has been known to occur before 20 and after 60. It has been said to be much more common in this country than in some others, such as America and Germany, and this may be the case; but it must be remembered that it is a comparatively rare disease, and hence the collected statistics are small, while cases have been especially well recognised and keenly sought for in this country during the short period of years that myxœdema has attracted clinical interest.

Morbid anatomy and pathology.—The thyroid gland is usually small, but may be enlarged by the presence of solid tumours or cysts, or excess of fibrous tissue. In all cases, however, there is partial or complete atrophy of the secreting structures, and in most a great increase of the fibrous tissue, which gives the gland a tough feel and a yellowish white colour. The other characteristic change is found in the subcutaneous connective tissue, the fibres and cells of which are increased in amount, and the spaces filled up with a transparent gelatinous material. Ord regards this substance as "a mucin-yielding modification or infiltration of the connective tissue," a view which has not been wholly accepted, owing to the infiltrating material being absent in some cases which have been examined, but he suggests that this may have been due to absorption previous to death in chronic cases, in which fibrous tissue only is found. The pressure of this substance on the hair follicles, sweat, and sebaceous glands gives rise to the loss of hair, and to the dry, atrophic condition of the skin.

All the other changes are inconstant. An increase of connective tissue has been found in the nervous system generally and in the principal viscera, including the ovaries and arterioles, but this may be absent, and if the records be accurate it varies greatly in amount and distribution when it is present. Slight degeneration of the cortical brain cells has also been observed, but not differing from what is found in many other conditions. The pressure exercised by the new growth of connective tissue will no doubt interfere with function, and accounts for certain of the symptoms of the disease. Enlargement of the thymus gland, and of the pituitary body, has also been recorded, but the significance of this is obscure.

In 1859, and again in 1884, Schiff had removed the thyroid gland in dogs, and found that they soon became subject to tetanic attacks, fell into a condition of emaciation and apathy, and finally died. Transplantation of the thyroid gland from another dog prevented these symptoms. Eiselsberg confirmed these results in carnivora, but found that herbivora fell into a chronic cachectic condition. Horsley removed the thyroid gland in monkeys, and describes them as suffering from a condition closely resembling myxœdema. He also found mucin in the blood of these animals, and plentifully in the secretion of the parotid gland. Hermann Munk, however (1897), denies the value of these experiments, which he repeated, and concludes that many of the animals remain in good health after thyroidectomy. There can be no doubt that light has still to be thrown on this part of the subject, but it is equally certain that in man removal of the thyroid gland gives rise to the symptoms of myxœdema.

In health the gland produces a colloid secretion, which it pours into the circulation, and which is necessary for the healthy nutrition and metabolism of the body. When this secretion is absent or defective, the changes seen in myxœdema result. We have no knowledge of its mode of action. Another view, not so generally held, is that the gland separates certain poisonous substances from the blood. The active substance in the secretion is an organic iodine-containing compound, which was named iodothyrin by its discoverers, Baumann and Roos (1895). The sheep's thyroid contains about 0·2 to 0·5 per cent. of this body, which is very stable, and is not broken up by the digestive processes, and hence the activity of the gland when given by the mouth.

Symptoms.—As a rule, myxœdema begins insidiously, progresses slowly, and may take many years to reach a height. In post-operative cases the earliest symptoms have been observed in from four weeks to a year, but many cases proceed much more slowly. Occasionally the progress is rapid, all the symptoms being fully developed in a few months. The first of these are subjective, and consist in a feeling of feebleness, tiredness, and coldness. The skin begins to get rather dry, and gradually the deposit of solid œdema can be detected under it. It is earliest observed in the face, which gradually acquires a stupid, heavy, apathetic, swollen look. All the features become coarsened, swollen, and enlarged; the lower eyelids have a baggy look, the upper eyelids are swollen and hang down over the eyeballs, the cheeks are pendulous, the nose is broadened, and the lips and chin coarsened. The skin has a waxy yellowish tint, and there is very often hyperæmia over the malar bones and nose. This, and a cyanotic tint of the lips, is often very characteristic. The hair of the scalp and eyebrows falls out, and this further accentuates the peculiar facies. The subcutaneous swelling gradually spreads to the root of the neck, and appears in the abdomen, hands, feet, and other parts, giving the person a clumsy, bulky appearance. The gait is deliberate, waddling, and awkward, and no movement of any kind is performed nimbly or neatly. The hands become more square in shape, or "spade like," owing to the fingers being swollen and non-tapering, and they are used clumsily; the feet also become swollen, and larger gloves and boots are required. The soft tissues of the mouth, throat, and larynx also become infiltrated, hence the tongue is enlarged, swallowing may be slightly interfered with, and the tones of the voice become thick, harsh, and monotonous.

The skin is very dry, owing to the lessening of the sweat and sebaceous secretions. It becomes coarse, much thickened, cracked at places, and is constantly scaling or peeling off. The surface has a cold feel, scarcely pits on pressure, and is inelastic and resistant to the touch. The hair, nails, and teeth all suffer greatly from malnutrition, the two first becoming dry and brittle, and the last carious or loose. The temperature is subnormal, usually 97° or 98° F., and undergoes little diurnal variation. The patient feels always cold, and is very susceptible to climatic conditions, becoming worse almost invariably in cold weather. There is marked muscular feebleness. The nervous system is very distinctly affected. Cerebration is slow, there is great deliberation both in speech and movement, and a disinclination for mental and bodily exertion. Memory becomes weakened, ideas are very slowly grasped and responded to, and there is sometimes an overpowering tendency to sleep at odd times. At night sleep is usually good and undisturbed. Both sensory and motor nerves are depressed in function, and the reflexes are somewhat diminished in activity. One or all of the special senses may be dulled. Most patients are contented and good-natured, not to say apathetic, but occasionally a morose, unsocial temper is developed, or depression, delusions, and mental alienation may ensue. A certain degree of anæmia always becomes developed, the corpuscles and hæmoglobin falling to about 60 or 70 per cent. of the normal. Bleeding from mucous membranes is not uncommon, and in women menorrhagia is frequent. Amenorrhœa is also not uncommon. The sexual instinct becomes greatly weakened, or abolished.

The pulse is a little slower than normal in most cases, and the heart's action tends to be feeble. Fainting and palpitation are not uncommon. Appetite and digestion are not vigorous, and constipation is often very troublesome. The urine as a rule is normal; the urea tends to be small in amount, and occasionally albumin, sugar, and mucin have been found in it. If cirrhosis of the kidney be present, there may be serous œdema along with the solid infiltration.

Even when untreated, cases of myxœdema live for many years and often to a good old age. Death may occur from exhaustion and the general malnutrition of the disease, but is generally due to intercurrent complications, such as the commoner lung, kidney, and circulatory affections.

Diagnosis.—In a fully developed case there is no difficulty, as the diagnosis is readily made from the appearance, symptoms, and history of the patient. In slight cases, in which there may only be a suspicion of myxœdema, the doubt may be resolved by careful treatment with thyroid gland. Myxœdema has also been mistaken for Bright's disease, anæmia, and corpulence, but the distinction is readily enough made by any one who is well acquainted with the clinical features of the disease.

Prognosis.—In most cases the prognosis is good, if thyroid treatment can be carried out systematically and continued. Cases of cure have been reported, but this can only happen where the gland has not been totally destroyed or removed, and where the portion left has become active and furnished a sufficient secretion for the needs of the body. If the patient be too old, or if the heart, arteries, or kidneys be affected, the results of treatment may be a little disappointing, but considerable improvement is invariable.

Treatment.—The treatment consists in supplying the active constituent of the thyroid gland to the patient. The most convenient preparation is the sheep's thyroid, dried, powdered, and deprived of its fat (*Thyroideum siccum*, B.P., dose, 3 to 10 gr.), given as a powder, cachet, pill, or tablet. One should begin with a dose of 3 to 5 gr. once daily, which may be increased or diminished according to its effect on the patient. There is an improvement in the subjective feelings within twenty-four or forty-eight hours. In a few days the subcutaneous swelling begins to disappear, and the whole aspect and demeanour are improved. Weight decreases rapidly, mucin often appears in the urine, which increases in amount and contains more solids. The skin grows soft and begins to secrete sweat and perspiration, the hair grows in, mental and physical activity return; and, in a word, after some weeks of treatment, the patient resumes his or her normal appearance. To maintain this, thyroid gland must be administered regularly. The amount required varies, and can only be ascertained in each case by experience, but a dose of 5 gr. twice a week, or oftener, suffices in most cases to keep the individual in a healthy condition.

Healthy persons are comparatively insusceptible to the action of thyroid, but in myxœdema unpleasant and even dangerous symptoms are very readily produced. The signs of an over-dose are occipital headache and pains in the body generally. Sometimes rashes appear on the skin. The most dangerous effect is cardiac depression, the results of which vary from slight faintness to complete collapse and death. If the heart is feeble or diseased, great care is necessary. Nervous excitement, diarrhœa, vomiting, and rarely glycosuria and albuminuria may be caused.

CRETINISM.

Sporadic cretinism is the name given to cases of cretinism occurring in localities in which the disease is not endemic. It is a comparatively rare condition, and is myxœdema in the infant. It is due to atrophy or absence of the thyroid gland, the results of which are much more severe than in the adult, owing to the complete arrest of physical and mental development at an early stage of growth. In some cases the gland seems to be congenitally absent, in others it has become atrophied during intra-uterine life, or some time after birth. In all cases the cause of this is obscure, although it has been attributed to syphilis, alcoholism, consanguinity of parents, maternal impressions, all of which are very common conditions, and yet rarely productive of cretinous offspring.

A typical cretin is stunted in growth and ill-proportioned, the head, hands, and feet being usually large, out of proportion to the rest of the body. The face is broad, the lips coarse, the nose flat, the tongue swollen, and the abdomen prominent and overhanging. The skin is coarse, thick, inelastic to the feel, and often very dry. The hair is scanty. Nutrition is poor, the temperature is subnormal, and the individual is very susceptible to cold. There is complete absence of sexual instinct as they grow older, and they vary considerably in the amount of bodily growth. As regards their mental condition, some remain completely imbecile, requiring to be fed and tended like infants, others can be educated into cleanly and simple industrious habits.

Thyroid treatment effects an improvement, which has sometimes been very striking and apparently complete. In other cases the results have been less hopeful. Sufficient time has scarcely elapsed to enable a definite pronouncement to be made as to the ultimate value of treatment in such cases.

Endemic cretinism is met with in the great mountainous regions of the globe, such as Switzerland, the Himalayas, Thibet, Pyrenees, etc. It is closely associated with the prevalence of goitre in these localities, and has been attributed to the water, soil, habits of life, and other causes. The true cause is unknown, but there is a good deal of evidence to show that it is something contained in the drinking water. The thyroid gland is in a condition of atrophy from early years or from birth, and this may give rise to a condition of helpless idiocy of the lowest type, or to slight idiocy, with stunting of growth, or to intermediate conditions of mental deficiency and bodily deformity. The treatment and training are conducted on the same principles as experience has shown to be useful in the case of idiots generally.

RALPH STOCKMAN.

DISEASES OF THE SUPRARENAL BODIES.

ADDISON'S DISEASE.

This is a chronic morbid condition, following on various lesions of the suprarenal glands, and accompanied by a deep pigmentation of the skin and sometimes of mucous membranes, by great general weakness, often by severe gastric symptoms, and terminating fatally.

History.—Single cases had been occasionally reported, as by Bright, Addison himself, and others; but any general interest in the disease was first aroused by the publication of Addison's book on "Disease of the Suprarenal Capsules" (1855), in which he described eleven cases, and attributed their occurrence to lesions of the suprarenal glands. Later, Wilks and Greenhow enunciated the view that the semilunar ganglia and sympathetic plexus became involved by disease affecting the neighbouring suprarenal bodies, and that the various symptoms followed in consequence of the nervous implication. More recent observation, however, does not confirm this view. Addison's clinical and pathological description was so complete, that since his time little, except as regards details, has been added to our knowledge in these respects; but within very recent years much physiological research on the nature and function of the glands has been undertaken, and this has resulted in the acquisition of a certain amount of definite knowledge, and in a very great deal of theorising based on somewhat slender foundations.

Etiology.—We only know that it follows on various lesions of the suprarenal glands. What determines some of these lesions is unknown; in other cases they are plainly due to traumatism, new growths, and pressure from different causes. Men are more commonly affected than women, and especially from 20 to 40 or 50 years of age, but cases at other ages have been recorded not infrequently.

Morbid anatomy and pathology.—By far the commonest lesion found is the so-called fibro-caseous degeneration of the glands, both being usually affected. It is a tuberculous change, tubercles forming primarily or secondarily in the medulla of the gland, then caseating, and often softening. During the process the irritation causes a large development of fibrous tissue. It then forms a large irregular mass, made up of fibrous tissue and soft caseous matter. Atrophy of the gland is not very uncommon.

Malignant disease, pressure from an aneurysm, or caries of the spine, and hæmorrhage are very rare as causes. Some cases have been recorded in which the glands were found unaffected, but the neighbouring sympathetic nerves and plexuses were involved by chronic inflammation or pressure. It is supposed that the blood and lymph supply of the glands must have been interfered with in these cases, and that this amounted practically to stoppage of their function. The pigment is simply an exaggeration of the normal skin pigmentation, and resembles what may be seen in very swarthy persons or negroes. It is deposited in the rete Malpighi, and in the rete mucosum of mucous membranes.

The theory that the symptoms of Addison's disease are due to irritation of the sympathetic nervous system has now only a historical interest, as they undoubtedly depend on abolition of the gland function. We are quite ignorant as to how this brings about the pigmentation, and we can only offer surmises regarding some of the other symptoms. It has been supposed that the glands have the function of destroying toxic substances, generated in the tissues; but Abelous and Langlois, after destroying the glands in animals, found that injection of suprarenal extract kept them in good health, otherwise they died in from four to thirty-six months with symptoms analogous to those of Addison's disease in man. It has been shown that the glands contain a substance which is a very active stimulant and raises the blood pressure (Oliver and Schäfer), and that this body is absent in diseased glands. Abel has isolated it in an almost pure state, and describes it as an alkaloid. The skeletal muscles are also stimulated and kept in tone by it, and this may explain the extreme muscular and cardiac weakness in disease of the glands.

Symptoms.—Addison's disease begins as a rule very insidiously, with gradually increasing weakness, which may slowly or rapidly develop into extreme asthenia. There is slight or marked muscular feebleness, along with great disinclination for exertion of any kind. The heart's action also becomes feeble; soft blowing murmurs may develop. When the disease has progressed, faintness is very common and fatal syncope not infrequently occurs, the pulse tending all through the illness to be small and weak. If the patient undergo any exertion, he suffers from dyspnœa, and the after-fatigue is so extreme that it often prostrates him for days. The feebleness of the circulation accounts probably for the slightly subnormal temperature and for the coldness of the extremities, which are commonly experienced. There is a complete want of bodily and mental staying power, especially under any strain.

The pigmentation of the skin probably develops later than the general symptoms first described. In a few cases it is entirely absent, but this is said to be due to death occurring early from tubercle or malignant disease. Sometimes the pigment is so slight that it is very difficult to make up one's mind that there is any very definite change in the colour of the skin; and from this it passes through all gradations, from a sallow yellowish tinge up to a deep greeny bronze. It begins in patches or as a diffuse yellowish greyness, which very gradually deepens and may affect the whole skin, but is usually in patches. The naturally pigmented parts, such as the axillæ, nipples, and genitals, are earliest and often most deeply affected. The face, neck, and back of the hands are also early affected, as is the skin over the joints and bony prominences, or where slight irritation and pressure are exercised by braces, or garters, or the clothing. Occasionally the pigment is arranged in circles, and then the darker patches contrast often very forcibly with the less deeply pigmented. Sometimes patches of leucoderma stand out very prominently. The nails, palms of the hands, and soles of the feet are seldom discoloured, but I have seen slight darkening of these parts, especially in the depressed lines of the skin, and also small, intensely dark deposits. The mucous membranes of the mouth and vagina are also very frequently involved. On the inner surface of the lips the pigment is usually in patches, determined by the pressure of teeth, but is also commonly enough found on the gums and on the roof of the mouth where there is no pressure. Rarely there is also a dark deposit in patches on some of the serous membranes, and the hair is often of a dull bronzed colour.

At the beginning of the illness the appetite may be poor, but there is often no especial dyspepsia. When it is thoroughly developed, however, severe indigestion and constipation, with repugnance to food, and obstinate and severe vomiting, become prominent and distressing symptoms. An attack of diarrhœa often proves fatal. There is probably a loss of tone and power in the muscle of the whole gastro-intestinal tract, and vague abdominal and lumbar pains are often complained of.

There is general nervous atonicity with depression and apathy. No pigment or other abnormal substances have been found in the urine. The blood corpuscles and hæmoglobin are not directly affected. There may be a certain amount of anæmia in some cases due to tuberculosis or malignant affections, but *per se* disease of the suprarenal glands does not seem to have any influence in this direction. In one case in which I examined the blood repeatedly, the red corpuscles were as high as 5,800,000 per cubic mm., and the hæmoglobin 96 per cent.; and in two cases which afterwards proved fatal from tuberculous disease, they were only slightly under the normal. Addison's disease is distinctly rare, without being very uncommon. Its duration varies from a few weeks to eight or ten years, but death generally ensues in one or two years after it has been definitely recognised. The duration, however, depends greatly on the cause, as the latter may be in itself fatal. The immediate cause of death may be exhaustion, syncope, diarrhœa, etc., and it may occur in coma or sometimes in a convulsive seizure.

Diagnosis and prognosis.—The diagnosis only presents difficulty in the early stages, when it is sometimes very hard to give a positive opinion. Abdominal diseases of different kinds, pregnancy, melanæmia, tinea versicolor, chronic phtheiriasis, and numerous other conditions, cause often an increase in the pigment of the skin, but they are easily enough differentiated. Where no pigmentation is present, lesions of the suprarenal glands are difficult to diagnose. The extreme weakness and the accompanying symptoms often give a clue. The outlook for the patient is always gloomy. The downward course is often very irregular, and periods of improvement intervene.

Treatment.—Great general care must be taken as regards exercise, dieting, and exposure to cold or damp, or any depressing bodily and mental influences. The patient should be kept in bed only if the exhaustion be great and the heart be feeble. Alcohol should be added to the diet, and any digestive disturbances or diarrhœa at once treated. Bismuth is perhaps the best means of controlling the diarrhœa. Ordinary tonic treatment with arsenic, iron, strychnine, bitters, cod-liver oil, etc., does not do much good, but has a certain value.

Latterly, treatment by administering suprarenal glands and extracts has attracted much attention. Oliver, Rolleston, Lloyd Jones, Osler, and others have reported improvement in the general condition under their use. Sometimes the pigment is lessened, sometimes not. In two cases where the lesion was tubercuIous I did not see the slightest improvement under their use, but in a third, where there was probably atrophy of the glands, very great amelioration in the symptoms and lessening of the pigment resulted after two months' treatment with three to six sheep's capsules daily, taken raw. On omitting the treatment the patient relapsed, and improved when it was resumed. He is still living and in fair health. Sansom has observed a somewhat similar case. It must be noted, however, that as yet no case has been reported in which complete and permanent recovery has taken place, although the subjective and objective symptoms are often very materially improved. Cases due to fibro-caseous degeneration seem to derive no benefit. The question of treatment by gland administration is still in a very unsettled condition.

RALPH STOCKMAN.

THE SPLEEN.

VARIOUS MORBID CONDITIONS.

Anatomically the spleen consists very largely of lymphoid tissue. Its capillary blood vessels are peculiarly developed to form wide spaces, an arrangement which gives rise to a very slow circulation in the organ, and to the constant presence of a large quantity of blood in it. Our knowledge of its physiological functions is in a highly indefinite condition. When the spleen is removed from a man or animal, death does not necessarily follow in consequence, its functions being apparently taken up by the other lymphoid structures of the body, which hypertrophy after its removal. It has been assumed that its function is the manufacture of red blood corpuscles, but this has been disputed on good grounds. It has also been supposed that it is actively engaged in the destruction of the red corpuscles, but for this belief there is also no convincing evidence. So far as our present knowledge goes, the spleen does not produce any special secretion, as some of the other ductless glands do, and my own observations have led me to the conclusion that spleen pulp and preparations made from it exert no visible action either in healthy or diseased conditions of the organ.

It is sometimes congenitally absent without apparently affecting the general health. Apart from injuries, it is doubtful if the spleen is ever primarily affected by disease, but it shares to an unusual extent in the effects of general morbid conditions, a result most probably of its anatomical structure and large blood supply. Foreign substances of all kinds circulating in the blood tend to be deposited in the spleen, owing to the slow blood current, and to the large number of leucocytes which it ordinarily contains. It is possible that the spleen may be the starting-point of certain of the so-called blood diseases, but proof of this is still wanting. At present, therefore, it is impossible to talk of diseases of the spleen. We can only enumerate the morbid conditions to which it is most frequently subject. These often give rise to no special symptoms, and very frequently cannot be detected during life, but often they cause more or less enlargement.

Perisplenitis is not uncommon in the course of other diseases, giving rise to local pain, tenderness on pressure, and friction. These symptoms are treated on general principles, namely, opiates internally, and hot or soothing applications externally.

The spleen is enlarged in many infective conditions, such as enteric fever, relapsing fever, scarlet fever, pneumonia, ulcerative endocarditis, anthrax, and pyæmia; in waxy disease, carcinoma, rickets, chronic syphilis; in splenic anæmia, acute and chronic malaria, leukæmia, lymphadenoma, general tuberculosis, and sometimes in pernicious anæmia. It is also enlarged in chronic venous congestion, from cardiac or hepatic disease or other causes, while hæmorrhages into its substance are not uncommon. A splenic cyst or abscess may also greatly increase its size and area of dulness to percussion.

The extent, character, and clinical features of the enlargement having been described under these different diseases, it is unnecessary to discuss them further here.

The spleen is found somewhat atrophied in chronic wasting diseases, and is occasionally so loosely kept in position by its attachments as to constitute the so-called "wandering" spleen.

RALPH STOCKMAN.

THE PITUITARY GLAND.

ACROMEGALY.

Syn., Marie's Disease.

A CHRONIC disease, probably connected with changes in the pituitary body, characterised by marked enlargement of the hands, feet, and face, enlargement of the bones generally, often some degree of general cachexia, with widespread but inconstant changes in nutrition and function.

History.—Pierre Marie described in 1886 two cases of this disease which he had observed in Charcot's clinique at the Salpêtrière, and to which he gave the name acromegaly, from its most obvious and striking symptom (*ἄϰρον* end, *μέγας* large). From these and five other cases, which he rescued from oblivion in the older literature, he was able to put on record the essential and most important clinical characteristics of the malady. In 1888 and 1889 he gave further and more complete descriptions, and in 1890, Souza-Leitz, his pupil, published a monograph on the subject, with accounts of thirty-eight cases collected from various sources. This work, along with Marie's original communication, has been translated into English by the New Sydenham Society, as has also been Sternberg's more recent monograph, which gives a very full bibliography up to the year 1898.

Etiology.—The pituitary body has been found to be the seat of pathological change—tumours, atrophy, hypertrophy—in all the more recent cases which have been subjected to post-mortem examination. Similar changes may be present, however, without acromegaly, and in the present state of ignorance regarding the functions of the pituitary body it is impossible to trace the steps in the causal connection. Heredity, severe illness, shock, and trauma have been noted as important in certain cases, but their influence cannot be regarded as more than predisposing. It most commonly commences during adolescence or early adult life, but cases occurring earlier and later are not very uncommon. It is a very rare disease on the whole.

Pathology and morbid anatomy.—The curious changes in growth are probably connected with an alteration in the functions of the pituitary body. As we do not know what these functions are, we can only say that they must have a close reference to bodily nutrition. The enlarged bones are coarser in all their parts, the air sinuses are enlarged, and so are all the roughnesses and promontories. The tissues of the skin are greatly hypertrophied, and the subcutaneous fat and connective tissue are increased in amount. Increase of connective tissue and degenerated cells have been described in all parts of the nervous system. Many other slight changes have been found post-mortem in individual cases, but as their relationships to acromegaly are by no means established, it seems unnecessary to enumerate them.

Symptoms.—Acromegaly usually pursues a very chronic course, lasting for many years, and commencing so insidiously that the typical bodily changes may be considerably advanced before attention is attracted to them. Certain cases, however, where a rapidly growing tumour involves the pituitary body, have been much more acute—three or four years in duration. Various functional disturbances may form the first subject of complaint on the patient's part, but the typical symptoms are connected with marked changes in the growth of certain parts of the body, especially the hands, feet, and head. The first indication that anything is wrong may be derived from the increased size of gloves, boots, or hat required by the patient. When the disease has fairly developed, one finds a gradual increase in the size of the hands and feet, which involves all the tissues and very seldom extends beyond the wrist and ankle. There is no deformity in the affected parts themselves, but simply a general increase of size, especially in breadth, all the tissues sharing in the hypertrophy, so that the extremities contrast very forcibly in their size with the proximal parts of the limbs. The bones of the cranium also become somewhat larger and denser, but those of the face are particularly affected. The malar bones, the frontal sinuses, the orbital ridges, and other prominences become very much enlarged; the lower jaw increases greatly in size and projects beyond the upper, so that the outline of the face is elongated and oval, and slightly concave. The forehead appears relatively small and low. The nose is enlarged in all its parts, the septum is thickened, the lips are swollen, especially the lower, which becomes turned down and overhanging, and the chin is large, massive, and well covered with flesh. The eyelids are thickened and swollen, the eyes look small in the large face, and often project somewhat. Add to this, that the skin of the face is often very coarse and pigmented, while the expression is dull and heavy. The vertebræ, sternum, clavicles, and ribs also hypertrophy. A very characteristic change is the occurrence of a cervico-dorsal curvature, resulting in a posterior hump, while a smaller projection is formed anteriorly by the enlarged sternum and ends of the ribs. The shoulders are rounded. The neck is short and thickened, the chin is usually allowed to rest on the chest; and all these changes together give rise to an attitude which has been compared to that of the anthropoid apes. The long bones and pelvis, although somewhat enlarged, are not usually affected to anything like the extent seen in other parts of the bony skeleton. The tongue is often very large, the hard and soft structures in the mouth may be greatly hypertrophied, and similar alterations in the larynx give the voice a rough, deep tone.

In the early stages of the disease the muscles are often well developed and powerful, but later, along with the supervention of general cachexia, they may become extremely feeble. Headache is often very severe, and may be constant or intermittent, but this, along with the giddiness, vomiting, disturbances of vision, and more rarely of hearing, merely indicates the presence of a cerebral tumour. The occurrence of bilateral temporal hemianopsia is a valuable diagnostic symptom of a tumour of the pituitary body, as it points to pressure on the optic chiasma. Drowsiness, severe insomnia, and general pains in the bones, may also be complained of. The skin is hypertrophied, is coarse in its texture, tends to fall into folds, and is very often covered with little pedunculated warty tumours. Polyuria, glycosuria, and peptonuria are all not infrequent, and are probably secondary to intracranial disturbances. Excessive thirst results in such cases. The appetite is usually good and often very large. Atrophy of the ovaries, uterus, and testicles commonly occurs, with more or less loss of sexual appetite, but the external genitals are enlarged in both sexes. Amenorrhœa is often a very early symptom. The heart is hypertrophied, the arteries are usually hard, and many of the veins varicose. In many cases the circulation is rather feeble, and a tendency to excessive perspiration on slight exertion is very frequently present. The thyroid, thymus, and pineal glands have been found hypertrophied and also atrophied; but the exact significance and importance of these, as well as of other observations made in cases of acromegaly, cannot be at present determined. The disease may last ten, twenty, thirty, or more years, and death may result from intercurrent disease or from gradually increasing cachexia and general debility.

Diagnosis and prognosis.—The anatomical changes and general symptoms make the diagnosis fairly easy in most cases. Acromegaly has occasionally been confounded with hypertrophic pulmonary osteo-arthropathy. In the latter, however, there is a history of chronic pulmonary disease, the hands and feet only are enlarged, the fingers are clubbed, and the typical facies and attitude of acromegaly are absent. The great chronicity of the affection makes the prognosis good as regards life, but as regards recovery it is hopeless. Certain cases seem to become stationary.

Treatment.—There is no specific treatment for acromegaly. The administration of pituitary, thyroid, and thymus glands has little effect on the course of the malady; improvement has been reported to have occurred in some cases, but occasional remissions are very common, quite apart from treatment, and hence great caution is necessary in forming an opinion. Resolvents, such as iodide of potassium and mercury, may be cautiously given in suitable cases. For the most part we are confined to the treatment of symptoms, such as the headache, sweating, neuralgic pains, and glycosuria, and this is done by means of the usual remedies.

RALPH STOCKMAN.

SECTION VI.

CIRCULATORY SYSTEM.

DISEASES OF THE PERICARDIUM.

The pericardium is subject to affections similar to those which are found in the case of other serous membranes, but modified by the special conditions which belong to it, so that there are differences in the results produced, as compared with the cerebral, pleural, or peritoneal membranes.

PERICARDITIS.

This affection presents several varieties. It may consist essentially in a fibrinous exudation, accompanied by comparatively little serum. There may, on the other hand, be but little fibrinous deposit, while the serous exudation is copious. The process may terminate by the emigration of a large number of leucocytes, so as to give rise to a purulent exudation. The exudate may further be of a hæmorrhagic nature, and, in addition to these various types, pericarditis may rest upon a special basis, such as a tuberculous or carcinomatous invasion.

Etiology.—New methods of investigation and modern modes of thought have entirely revolutionised this aspect of the subject, and have led to the universal recognition that such a conception as idiopathic pericarditis is untenable. When all the facts belonging to any supposed instance of the kind are candidly investigated, it is invariably found to have some relation to circumstances which fully explain its incidence.

Predisposing causes.—The disease is more common in men than in women, probably on account of the greater liability of the former to the primary affections from which pericarditis takes origin. It is more particularly an affection of early adult life, being comparatively rare in childhood and in advanced years. In old age, however, there is a somewhat increased liability to the disease, especially amongst those who suffer from certain diatheses. The effects of climate are considerable, no doubt in consequence of lessening the resistance of the tissues by impairing their vitality. Occupation and surroundings probably play some part in the evolution of the disease, when they are of such a character as to lead to diminished resisting power.

Exciting causes.—The determining factors in the production of pericarditis may be divided into the three groups of direct extension, infective processes, and constitutional diseases.

The disease may take its origin by extension from other parts of the heart, as in the case of endocarditis and myocarditis. On account of the close connection with the pleura and lung, it is a common experience to find that pleurisy and pneumonia are complicated by pericarditis. One fact must be remembered, however, in this connection: the source of the pericardial implication may be through organisms conveyed by the blood. Pericarditis produced by pneumococci has been seen without coexisting pneumonia. Suppurative processes in the mediastinal glands, and even in the lungs, have given origin to pericardial complications; while affections of the œsophagus sometimes, although much less commonly, produce the disease. In still rarer instances, affections of the bones and the thorax, *i.e.* the sternum and ribs in front, and the vertebræ behind, are causes of the affection.

All acute general diseases due to infective processes are capable of producing pericarditis. Some of these affections are comparatively frequent causes of the disease; the majority, however, are only exceptionally complicated by it. Scarlet fever, smallpox, erysipelas, influenza, septicæmia, and tuberculosis are the general diseases belonging to this category which most commonly give rise to pericarditis.

Amongst the diseases which are yet often termed constitutional—although certain are without doubt due to microbic influences—and which exert a determining influence in the evolution of pericarditis, rheumatism holds the first place. It is, in short, the cause of the overwhelming majority of all cases of pericarditis. The largest collection of figures bearing upon the relations of the two diseases is that of Sibson. This accurate observer found that of 326 cases of acute rheumatism sixty-three suffered from pericarditis, a percentage of rather more than twenty. The pericardial implication usually appears during the second half of the first week of an attack of acute rheumatism. Sometimes the pericarditis precedes all other symptoms of rheumatism. Chorea is not uncommonly associated with the disease, and their coexistence denotes a rheumatic link. Gout occasionally leads to pericarditis, but very much more rarely than does rheumatism. Certain of the blood diseases may be the cause of pericarditis, chief amongst which are scorbutus and purpura.

Affections of the kidneys act in a manner analogous to those of the constitutional diseases by the retention within the system of faulty chemical products, yet it must be allowed that in certain instances, secondary infection and ptomaine poisoning may, as has been suggested by Hanot, be the direct causes of the complication. The percentage of cases of Bright's disease so involved, varies very widely according to different observers.

Direct injury sometimes produces pericarditis. There can be no doubt that this cause is operative by allowing the entrance of micro-organisms, yet it is possible that a sterile pericarditis may thus occur. It has long been known that the pressure of an aneurysm or a tumour may give rise to an adhesive form of pericarditis, a fact which throws some light upon the possibilities of pericarditis from injury.

Morbid anatomy.—Although there are many differences in detail, yet a somewhat similar course is run by pericarditis in all its forms.

Fibrinous pericarditis.—The earliest appearance in acute fibrinous pericarditis is dulness of the membrane, which becomes somewhat ruddy in colour, and often shows ramifying blood vessels along with small hæmorrhages. Upon the part so affected, a layer of fibrin is deposited. This occasionally presents an almost smooth surface, but much more usually it is rough. The inequalities of the surface are sometimes like ripple markings on the seashore, or the surface of a honeycomb, or the reticulum of the calf; even more frequently the appearance is shaggy, as if butter had been pressed between the dairy implements known as "Dutch hands" in a dry state, and they had afterwards been separated. A certain amount of fluid is usually present. This may be almost clear, or it may be opaque from the presence of leucocytes, or coloured by blood. Such are the appearances in what is commonly termed fibrinous pericarditis.

Serous pericarditis has features closely resembling those just described as regards the surface of the membrane, but it is characterised by the presence of a much larger quantity of fluid; the amount has reached 160 oz. In such cases the fluid may be clear and transparent, or turbid and opaque. The colour of the fluid exudation varies between a pale straw tint and a dirty brownish grey.

Microscopic examination of such cases shows that the fibrinous exudation consists of layers of fibrin felted together, like the strata of a schistose rock, in the meshes of which there are small round cells, leucocytes, and hæmocytes. An infiltration by small cells can be seen both in the epicardium and pericardium. In most cases masses of organisms may be found amongst the fibrinous network. At a somewhat later stage the fibrin is found to be undergoing organisation by the intrusion of capillaries into it, and this process may end by a simple thickening of the membrane,—the so-called milk spots, or maculæ tendineæ, —or the result may be more or less adherent pericardium.

Purulent pericarditis is usually connected with general septicæmia, or arises from some neighbouring suppuration. The surface of the membrane in such cases is often thick and fleecy; sometimes, however, the fibrinous deposit has disappeared entirely, and there may even be ulceration of the pericardium. The fluid contained by the sac is always opaque and of a greenish yellow colour. It is found, on microscopic examination, to contain a large quantity of leucocytes, with some red blood corpuscles, and flakes of fibrin. Some of the cells may be seen to have undergone fatty changes. Pyogenic organisms are usually found in large numbers, but sometimes the fluid is sterile, probably from the death and destruction of the microbes. In certain cases of purulent pericarditis the pus has undergone decomposition. This is especially found when the affection has arisen by continuity from a suppurating cavity exposed to external influences. The pus in such instances is marked by its offensive character.

Hæmorrhagic pericarditis may arise from some malignant change, as in cancer; some constitutional disorder, such as scorbutus; some acute infection, like smallpox; or the presence of an aneurysm weeping into the sac after irritation by previous pressure. It need hardly be added that small wounds of the pericardium may also give rise to this type of affection. A fibrinous deposit lies upon the surface of the membrane and usually contains a large number of hæmocytes. Both epicardium and pericardium are deeply stained, and extravasations are present on both. Occasionally the surface of the membrane is eroded. The amount of fluid is extremely variable, and the quantity of blood which it contains has wide limits. In an early stage the blood is coagulated, and no change is apparent in the characters of the red blood corpuscles. At a later period, however, the hæmocytes undergo decomposition, so that the colouring matter is set free and becomes diffused throughout the effusion. Microscopic examination shows that the deposit upon the membrane is composed of layers of fibrin embracing many hæmocytes within its meshes. It further shows that in the abraded portions, the endothelium, and even the subjacent elastic layer, have been destroyed. In the case of malignant invasion, characteristic structures may be found on microscopic examination.

Tuberculous pericarditis usually takes its origin in the formation of localised masses in the epicardium. It may also, however, but much less commonly, be only one aspect of general miliary tuberculosis. As most frequently seen, small nodules are present in the membrane, invading to some extent the underlying myocardium. These nodules are surrounded by fibrinous deposits and organising tissue, often characterised by the presence of giant cell systems.

The pathological results of pericarditis present a very variable series. The most satisfactory termination of sero-fibrinous pericarditis, and that which is the most common, consists in re-absorption of the exudation, and more or less perfect resolution. Thickenings of the pericardial membrane frequently result from organisation of the fibrinous deposit, and if the deposits on both epicardium and pericardium become united and organised, more or less obliteration of the sac occurs. Such adhesions sometimes extend beyond the pericardium, so as to involve attachments to the anterior chest wall, to the œsophagus and aorta behind, to the pleural sacs, lungs, and diaphragm laterally and below.

In suppurative pericarditis, perforation of the sac has been observed, leading to far-reaching complications.

Symptoms.—Since pericarditis undergoes development under so many different conditions, its clinical features are extremely diverse. It may, for instance, be so masked by the appearances of a primary affection, to which it is due, as to entirely escape detection, and it may, apart from such attendant circumstances, have such an insidious onset as to escape unnoticed. In the majority of instances, however, it presents features which command the investigation of the physician. Sometimes, although not commonly, an attack of pericarditis is heralded by rigors.

Fibrinous pericarditis. — The temperature is extremely variable. Although usually elevated, it is sometimes normal, or even subnormal; in fact it seems to be entirely dependent upon the nature of the affection to which the pericarditis owes its origin. If it occurs as a complication of an acute disease, it may be ushered in by a slight increase in the pyrexia previously observed. On the other hand, the temperature may undergo no alteration, or it may even fall. If pericarditis should take its origin during the decline, or after the disappearance of pyrexia in any disease, there is almost invariably a rise of temperature.

Sensations of discomfort are sometimes present. It has been known since the observations of Harvey, that the pericardium in a condition of health is almost destitute of sensibility, but when pericardial irritation is present, the excitability of the afferent nerves becomes greatly increased. The nature and extent of the sensory disturbances vary considerably. There is, for the most part, nothing more than a sensation of præcordial uneasiness, but it may be considerably greater, so as to produce a feeling of oppression, or even of actual pain. Occasionally the pain has a darting or radiating tendency. The position of the pain is usually over the heart, but it may be felt in the region of the epigastrium, or in the back below the left scapula, or between the shoulders, or, in rarer instances, about the attachments of the trapezius and sterno-mastoid muscles. Sometimes when the pain has a darting character, its distribution involves the left shoulder and arm, so that it comes to resemble that of angina pectoris. To complete this aspect of the subject, it may be added that when such subjective painful sensations are present, there is almost invariably hyperæsthesia over the region in which discomfort is felt. This may, indeed, be present, even in cases when the patient is unconscious of any discomfort.

Interference with the action of the diaphragm is sometimes observed, and leads to the discovery of pericarditis. It is obviously due to reflex action, and may or may not be associated with painful sensations. Pain on swallowing is sometimes observed. It was noticed by Morgagni, and many instances have since his epoch been recorded. Complaints are occasionally made of palpitation, or of sensations of trembling or fluttering of the heart.

Breathlessness may be developed in every variety of pericarditis, but it is not common in the fibrinous or plastic type. It may, however, appear even in this form of the disease, when it may be of reflex origin, or caused by wide-spread adhesions to the pleuræ and chest walls. In cases marked by a considerable amount of fluid exudation, or if the pericarditic process has set up a paralytic myocarditis, the dyspnœa is produced by interference with the action of the heart.

All these symptoms are entirely subordinate to, and overshadowed by, the results of physical examination. This is not merely the case in such instances as are latent, but also in such as have definite rational symptoms. The pulse yields evidences of considerable alteration. During the onset of the affection, the pulse rate is usually augmented to a considerable extent, and the ordinary ratio of the pulse and the respiration undergoes alteration, since, with considerable acceleration of the former, there is but little alteration in the latter. The increase in the rate of the pulse is not at first attended by any noteworthy change in its other features, but after a longer or shorter time the vessel becomes empty, the pressure falls, a tendency to dicrotism appears, and the pulse is often irregular. It is probable that some of these appearances are produced by an extension of the morbid process to the myocardium.

The examination of the chest does not yield any decided information to the eye, except in the case of very thin patients. In such cases, the cardiac impulse may be at first somewhat more violent than in health. There is never any alteration in the form of the chest. Palpation at the outset of the disease sometimes gives evidence of increased force of the cardiac impulse. This is not of long duration, and is more or less speedily followed by diminution of the intensity of pulsation. In the later stages, the beat is felt to be somewhat slow and sluggish; occasionally it gives rise to a feeling as if the heart crept below the hand. There is, in addition, another important physical sign furnished by the hand—a trembling vibration may be produced by the friction between the epicardium and pericardium. When present, it as a rule only occurs with systole, and does not accompany diastole, but in rarer instances it is found with both. The most important point in regard to it is that it does not accurately coincide with the cardiac movements, but follows them.

Percussion shows that in the early stages of the disease there is no alteration of the cardiac dulness, but after the expiry of a few days it is sometimes found to be slightly increased on account of some dilatation.

The most important physical signs in pericarditis are those furnished by auscultation. The results of auscultation consist in two classes of clinical features—alterations in the sounds of the heart, and accompaniments produced by the pericardial change. The most common alteration of the heart sounds is a simple reduction in their intensity, with an increase in their frequency. The weakening of the sounds may in some instances be the result of reflex causes, but it is much more likely that it is due to myocarditis, arising by extension from the pericardial change. The first sound is often doubled, probably on account of some weakening of the myocardium, whereby the perfection of its contraction is somewhat impaired, that side of the heart most implicated being somewhat slower in producing the pressure necessary for the greatest intensity of the sound generated by it. The most characteristic of all the physical phenomena in pericarditis is the production of the friction sound. This appearance, described but misunderstood by Laennec, has been known since the investigations of Collin to be almost pathognomonic of pericarditis. It is perfectly true that the sound of friction over the præcordia may be produced by other causes of roughness than pericarditis, as for instance milk spots on the epicardium, and sclerosis of the coronary arteries. With a few such exceptions, nevertheless, friction heard over the præcordia, synchronous with the cardiac movements, is pathognomonic of pericarditis. The character of the friction murmur varies considerably. It may be of such a soft nature as to resemble the sound produced by rubbing silk or paper together, but when more intense, it is like the noise produced by the friction of two surfaces of rough leather, or even unpolished wood. It always conveys to the ear the impression of being superficial, and when the stethoscope is allowed to press somewhat heavily, its intensity is increased. This, however, is only true up to a certain extent, for if the pressure be too severe, the sound may cease altogether. The friction sound is only propagated to a very limited extent, and very rarely, if ever, passes beyond the limits of the præcordia. It is of interest to note that the intensity of the sound varies with the phase of respiration, but there is no certain rule in regard to this fact, the friction being sometimes louder when the chest is in the position of full inspiration, at other times of full expiration. Another point of importance is that the posture of the patient has considerable influence upon the intensity of the friction, which is usually heard with greatest distinctness when the patient sits up or leans forward. This is almost certainly a direct effect of gravitation. Gentle pressure with the stethoscope intensifies the friction, but if the pressure is excessive it may diminish or obliterate the sound. The rhythm of the friction sound presents certain differences. It can only, from its cause, be an accompaniment of the cardiac movements, and it differs from pleuritic friction by persisting when breathing is suspended, which gives an easy means of distinguishing between the two. Pericardial friction may accompany the systole, or both systole or diastole, while it is not infrequently found as a triple murmur, attendant upon auricular systole, ventricular systole, and ventricular diastole. In rarer instances, the sound is even more broken up, the systolic or diastolic portion being separated into several distinct portions. One point of some interest is that the friction sound does not absolutely coincide with the cardiac phases, but is almost always later. No doubt the reason of this lack of synchronism is analogous to what is found in pleural friction, since a certain amount of movement of the one surface upon the other must take place before sufficient friction is produced to generate the murmur. The duration of the friction is somewhat variable, but in simple fibrinous pericarditis it is rarely present for more than a few days, and it may indeed disappear after a very few hours have elapsed from the time of its onset. One interesting distinction in the course of the pericardial murmur, as contrasted with the pleural, is that the former usually persists even after serous effusion has occurred, unless this is present in considerable amount.

Serous pericarditis.—When the exudation of serous fluid takes place, some new clinical features make their appearance. The temperature usually undergoes little change, but if it has previously manifested a high range, it is, as a rule, somewhat common to observe a fall when the effusion takes place. If there has been pain during the onset of the affection, it almost invariably becomes less intense, and its place is taken by other sensations, which may, if pain has been previously absent, be the first subjective symptoms. Such sensations are generally vague, consisting in feelings of uneasiness, weight, oppression, or sinking. A tendency to syncope is by no means infrequent. The respiration becomes modified in certain definite directions. Persistent anhelation may be present, but this is often associated with paroxysms of dyspnœa, and the breathlessness may be so considerable as to reach orthopnœa. Such symptoms are more common when the effusion is at once large in amount and sudden in appearance.

Changes in the voice occasionally occur, probably in most instances from direct pressure upon the recurrent laryngeal nerve of the left side. It is only right to mention that the high authority of Sibson has been given in support of the view that an extension of the pericarditis to the recurrent laryngeal nerve is the cause of the aphonia. Hiccough sometimes results, and may be caused by pressure upon the phrenic nerve, or by mechanical interference with the diaphragm. Dysphagia is not uncommon. It is always worst when the effusion is most considerable, and it is rather easier in the sitting than in the recumbent posture.

The appearance of the patient presents no uniform characters, and it may be said that the complexion varies in accordance with two factors—the presence of pyrexia, and interference with the venous system. If febrile reaction is present, there may be a flush upon the cheeks of a bright or dusky tint. When there is interference with the return of blood to the heart, cyanosis may be seen, and this may be either general or local. It sometimes is only to be observed in the face and upper extremities. Œdema may be present, and it, like the cyanosis, may be confined to the face, head, neck, and upper extremities; more commonly however, it is general, and therefore to be seen mostly in the dependent parts. The renal secretion is usually scanty, of dark colour, and of high specific gravity. It may contain albumin, as the result of venous stasis.

The pulse becomes of less volume and lower pressure when the effusion is considerable, and there is a great tendency to dicrotism. It is also irregular as well as unequal in many instances, while the bigeminal, alternate, or even paradoxical pulse may be observed. Sometimes the left radial and carotid pulses are smaller than the right. The veins of the neck often show considerable distension, and distinct venous pulsation may be seen, which is usually produced by auricular systole.

Most of the symptoms which have thus far been referred to are the direct result of the increase of fluid in the pericardium, as was quite clearly demonstrated by Lower, more than two centuries ago, and the backward pressure is caused by the interference which the effusion causes with the action of the auricles.

There is sometimes, but by no means commonly, some fulness of the præcordia, more especially of the intercostal spaces along the left edge of the sternum. The cardiac impulse becomes much less distinct. On palpation these facts can be better appreciated. The apex beat is so thoroughly obscured in some instances as to be imperceptible by touch, and it is sometimes more mobile from the enlargement of the sac by fluid. Fluctuation has sometimes, but rarely, been found. Vocal fremitus over the præcordia becomes feebler. The area of cardiac dulness progressively increases during the process of fluid exudation, until it attains a maximum, from which it gradually diminishes. The increase takes place in every direction, and affects both the deeper or relative and superficial or absolute dulness. The form which is assumed by the dulness is, in cases of much effusion, eminently characteristic, and has been compared with greater or less aptness to different objects. The form is more like the outline of a pear hanging downwards by its stalk than anything else. The narrow upper portion of the cardiac dulness forms a peak, and where it joins the lower or broader portion there is an indentation at the left side. This characteristic form is often modified by pericardial adhesions, or by affections of the lungs, yet it is in many cases remarkably constant. In early effusion there may be, as Rotch has shown, some increased dulness in the fifth right intercostal space. The results of auscultation are almost entirely dependent upon the amount of exudation. The heart sounds always lose some of their intensity, partly from passing through the fluid medium, partly from interference with the aspiratory and expulsive functions of the heart, and partly, in at least a certain proportion of cases, from extension of the disease-process to the myocardium. Systolic murmurs, having their origin at the mitral and tricuspid orifices, are extremely common. In by far the larger proportion of cases the friction sound persists. It disappears in fact only in those cases in which the exudation is excessive. Although it persists, it must nevertheless be remembered that its intensity wanes.

The usual result of serous pericarditis is more or less complete resolution by reabsorption of the fluid. The fibrinous exudation is also for the most part removed, but a certain amount of it commonly undergoes organisation, and may even give rise to some adhesions. On the other hand, the contents of the sac occasionally undergo further morbid changes, giving rise to purulent pericarditis.

Purulent pericarditis.—The clinical features in this form of the disease differ according as pus formation is a primary process or has occurred in consequence of some alteration in a lesion already present. When the affection has from the first been attended by purulent exudation, as an incident, for instance, in the course of a septicæmic affection, there may be no alteration in the general symptoms, and the only possibility of recognising the pericardial complication lies in the presence of its physical signs. When the pericarditis is the principal result of an invasion by pyogenic organisms, the symptoms are more definite. The onset of distinct and recurring rigors with characteristic fluctuations of the temperature, and profuse as well as repeated perspirations, may give definite information of a general kind. The affection, however, has often been found with no alteration of temperature, and therefore such symptoms are not infallible. When an exudation of a purely serous character becomes purulent, it is easier to arrive at correct conclusions. In such an instance, rigors, followed by characteristic fluctuations of temperature and pulse, with excessive perspirations and hectic tendencies, will indicate the change which has occurred in the nature of the exudation. The pulse is almost invariably of extreme frequency; the vessel is empty, and the blood pressure is low; dicrotism and irregularity are common. There is diminution in the force of the cardiac impulse, increase in the area of cardiac dulness, diminution in the intensity of the cardiac sounds, and sometimes the presence of friction, just as in the case of serous pericarditis.

Hæmorrhagic pericarditis—In a certain proportion of cases the exudation contains some blood, more especially when it occurs in the course of an already existent cardiac or renal disease. In elderly patients, and in the presence of certain diathetic conditions, blood is also present. The clinical phenomena in such cases seldom present any features to be regarded as characteristic, but if there be any considerable degree of hæmorrhage it is revealed by symptoms resembling those produced by serious loss of blood in general. The presence of faintness and giddiness, with chilliness of the extremities, and cold, clammy perspiration, attended by irregularity and emptiness of the pulse, as well as great weakening of the cardiac impulse and sounds, may be sufficient to give evidence of the nature of the affection.

Chronic pericarditis.—In certain instances, one or other of the ordinary acute forms of pericarditis does not undergo resolution in the ordinary course, and remains as a chronic affection. In addition to this, there are undoubtedly instances which, from the first, are of a chronic type. This is more particularly the case in feeble and elderly people, and it is certainly more common in those who are alcoholic, or have suffered from renal disease, than in any others. When it follows upon an acute form of the disease, the clinical features are simply those of the stage which the affection has reached, and to which it is therefore unnecessary to refer further; but the disease is sometimes from the first chronic, in which case it pursues an entirely latent course. There is usually weakness of the circulation, with a tendency to cyanosis and dyspnœa. There may be some oppression in the præcordia, but there is very rarely any distinct pain. The pulse is empty, feeble, and irregular. There is weakness of the cardiac impulse, enlargement of the area of dulness, diminished intensity of the sounds, and there may be a friction murmur. In the absence of the latter physical sign, the condition may closely simulate hydro-pericardium.

Diagnosis.—The recognition of pericarditis does not usually present much difficulty, seeing that the physical examination furnishes unmistakable evidence. There are, however, one or two directions in which it is possible to err. Care must be exercised, in the first place, not to mistake pericardial friction for a similar sound generated in adjacent viscera. Pleuritic friction, especially when produced on the outer side of the pleural pericardium, may be mistaken for the friction of pericarditis, but it ceases when the respiratory movements are stopped. A similar friction sound is occasionally heard in cases of peritonitis, by the rubbing of the roughened surfaces during the respiratory movements of the abdomen, but it also necessarily ceases when the breath is held. It is scarcely necessary to mention in this place, that a friction sound has been heard in tuberculous peritonitis attending the movements of the heart in consequence of the rubbing together of the diaphragmatic peritoneum and that covering the liver. It is so rare, however, as scarcely to merit attention. Probably it is scarcely necessary again to revert to the fact that a friction murmur in the pericardium may be produced by dryness, milk spots, and roughness over the coronary arteries.

The friction sound may be mistaken for an endocardial murmur, but its superficial character and grating quality ought to serve for its recognition, while the facts that it is not propagated beyond the præcordia, and is increased in intensity on pressure, furnish additional aids in distinguishing it. The rhythm of the exocardial friction is also helpful, since, as has been previously mentioned, the friction sound always follows by some interval the cardiac movements.

In the case of a serous effusion, diagnostic difficulties increase, rather than diminish. The increased area of cardiac dulness may be mistaken for a dilated heart, or for hydro-pericardium, while mediastinal tumour and even thoracic aneurysm may be simulated. Consolidation of an adjacent portion of lung and encysted pleurisy may also be sources of error. In most instances, the conditions under which pericarditis arises will prevent the possibility of error, and it is only when cases present themselves without any distinct history of the mode of onset, that difficulties arise. Even in such cases the characteristic form of the area of dulness ought to be sufficient to differentiate pericarditis from mediastinal tumour, abscess, thoracic aneurysm, pulmonary consolidation, and encysted pleurisy. Nevertheless, some instances in which pericarditis is attended by alterations in the radial pulses, and differences in the pupils, may give rise to considerable doubt. Hydro-pericardium will, in almost every instance, be found to take its origin in causes which are obviously existent, while the physical signs of cardiac dilatation are usually unmistakable.

Prognosis.—The prospects in pericarditis are largely dependent upon factors belonging to the individual peculiarities of every case. It is more serious during childhood and old age than at other periods of life, and it is more fatal in women than in men. The powers of resistance of the individual have considerable influence over the future, and the existence of any previous affection, such as renal disease, by which the metabolic functions are retarded, or exhaustion, whether from excess or fatigue, necessarily renders the outlook less hopeful.

The severity of the affection which has produced pericarditis largely influences the prognosis. As an accompaniment of ordinary acute rheumatism, pericarditis in itself cannot be regarded as an affection of great severity, and the statement is even more true as regards most of the infectious diseases which it may complicate. When pericarditis is accompanied by endocarditis, the whole aspect of the case becomes more serious, and almost all fatal cases are due to the combination of the pericardial and endocardial lesions. If, in fibrinous pericarditis, there is also pleurisy, with much tendency to adhesions, the outlook is graver.

In serous pericarditis the amount of fluid plays the most important part in prognosis, while the nature of the fluid which is present is of even greater weight. Speaking generally, it may be said that the outlook is the graver the greater the amount of fluid, while the presence of purulent and hæmorrhagic effusions in the pericardium is most serious of all.

Treatment.—In certain affections, more particularly in acute rheumatism, which are liable to be complicated by pericarditis, questions of prophylaxis will necessarily arise. The specific treatment of acute rheumatism by means of the salicyl series of drugs has, so far as it is at present known, unfortunately not had the effect of greatly diminishing the various cardiac complications of the disease. In addition to persevering with the remedies already in use, it may be possible, by means of other methods of treatment, to obviate the tendency to pericardial implication. Two methods are of real utility. One of these consists in the application of cold over the præcordia by means of the ice-bag or of Leiter's tubes: the other, which is of greater efficacy, is the application of repeated small blisters to the chest wall and front of the heart. It is unnecessary to remark that under such circumstances the patient will without doubt be under general management for the disease already in existence.

After the appearance of acute pericarditis, the indications are to restrain the morbid process, and to remove its effects. The first of these may be met by keeping the cardiac energy at a level as low as possible. For this purpose absolute rest is necessary, and the patient should on no account whatsoever be allowed even to sit up in bed. Sleep is to be favoured as much as possible. The diet should consist chiefly of milk and soup, and the alimentary functions should be carefully watched. Painful sensations may be greatly relieved by the application of cold, but in certain cases, where there is much depression, heat is even more useful. The employment of two or three leeches applied to the præcordia often causes great relief. It sometimes happens that the distress is so great that an anodyne is necessary, and in most instances there can be no objection to the use of opium.

The medicinal remedies already in use before the onset of the pericarditis must be continued. If the temperature, in spite of the action of such remedies, has a tendency to become elevated, it will be advisable to give one of the modern antipyretics, the most useful of which is phenacetin. Aconite has sometimes been recommended, but it ought on no account to be given. There is only too great a tendency to a fall of arterial pressure from diminution of cardiac energy, and the aconite class of drugs should be absolutely shunned. An undue frequency of the pulse, or tendency to diminish arterial pressure, will point to the necessity for general stimulants. Alcohol in the form most agreeable to the patient will probably be sufficient for the purpose, but the ammoniacal and ethereal stimulants may be required. It is sometimes necessary to employ one of the cardiac tonics. If any considerable effusion takes place, a new aspect is given to the case. It is, however, unnecessary in most instances to make any alteration in the management already adopted, while sedulously watching for the development of any symptoms produced by fluid exudation. If there should be any evidence of pericardial distension, diuretics and purgatives will be advisable, along with counter-irritation externally; and at this stage the iodide of potassium may be employed along with saline diuretics.

When it can be ascertained with certainty that there is a pyogenic cause, the appropriate serum should be employed. It is sometimes possible to determine bacteriologically that the organisms are streptococci or pneumococci, and in such circumstances the antistreptococcic or antipneumococcic serum is indicated.

The question of operative interference sometimes arises when fluid effusion is considerable. The most useful means which can be adopted is the employment of the aspirator. Opinions differ widely as to the most suitable site for the operation, but it may be said that there are two positions least open to objection. One of these is in the fifth intercostal space just inside of the left mammary line; the other is in the angle between the ensiform cartilage and the costal margin. The statistics of Roberts, dealing with sixty cases of paracentesis pericardii, show that twenty-four ended in recovery, giving a mortality of 60 per cent. In cases which give features raising a suspicion that a purulent accumulation is present, the hypodermic syringe ought to be employed for diagnostic purposes, and if the suspicion be confirmed, the exudation should be removed. It may be possible to bring about recovery by means of aspiration, which may, however, require to be performed more than once, but in certain cases a free incision will be necessary.

ADHERENT PERICARDIUM.

Regarded by ancient writers as a congenital absence of the membrane, obliteration of the pericardial sac has only been understood since the observations of Vieussens.

Etiology.—Adhesions between the pericardial layers may probably be the result of every variety of pericarditis, but there is much greater tendency to it in the fibrinous than in any other type of the disease. The formation of adhesions may occur in an early stage of the affection before any fluid effusion has taken place, but it is probably more common to find that it takes place after the re-absorption of a fluid exudation. It is frequently associated with pleural adhesions, which speak for the wide extension of reaction processes. Statistics with regard to the incidence of this lesion give very conflicting results. According to some authors, adhesions occur in 5 per cent. of cases; according to others, they take place in nearly 50 per cent.

Morbid anatomy.—The extent of the adhesions is very variable, the extremes being the slightest attachment by means of slender fibrinous bands to complete obliteration of the sac. It is not at all uncommon to find fatty degeneration in the newly formed tissue, uniting the pericardial layers, and calcareous infiltration also occurs. The heart itself undergoes consecutive changes; in something like 90 per cent. of all cases of uncomplicated adherent pericardium, by which is meant pericardial adhesion without valvular disease, there is cardiac hypertrophy. The reason for this is probably not so much that the work of the heart is increased by the adhesions, as that these interfere with the complete emptying of the organ, so that a tendency to dilatation is produced, and the residual blood acts as a determining cause of hypertrophy.

Symptoms.—Adherent pericardium may run a course absolutely latent, in which case the affection is only discovered accidentally on examination, or may indeed be found after death. There may, on the contrary, be general symptoms of the kind common in cardiac disease, such as palpitation, cyanosis, breathlessness, and œdema. There can be no doubt of the accuracy of John Broadbent's statement, that the right side of the heart suffers more than the left. In some instances there is considerable uneasiness and distress in the præcordia, and the fact of sudden death is not infrequently found. The pulse may present many abnormal characters. It is often, however, irregular and unequal, while the paradoxical pulse—described by Kussmaul as pathognomonic of mediastinal pericarditis—not infrequently makes its appearance. During the diastole of the heart, a distinct collapse of the veins of the neck is often seen, following a systolic swelling. Sometimes the left portion of the præcordia is more prominent than the right, but of more importance are the movements communicated by the heart to the parietes. The apex beat is almost invariably somewhat more energetic than in absolutely healthy conditions, and it is commonly associated with systolic retraction of the surrounding region. The left half of the epigastrium is drawn in with each systole, and the intercostal spaces to the left of the apex beat are also pulled inwards. John Broadbent has pointed out that the posterior as well as lateral parts may be drawn in. The view expressed by Skoda, that a drawing in of the apex occurs during systole, is quite untenable, both from the physiological and clinical point of view. During the diastole, a well-marked rebound takes place, and constitutes the most characteristic feature of adherent pericardium. All these facts can be observed both by inspection and palpation, but they are necessarily more patent by the latter method. When a change of posture takes place, the apex-beat is found not to travel so freely as in healthy conditions. The inspiratory movements of the central portion of the abdomen may be lessened, and during inspiration a retraction of the lower portion of the sternum is occasionally seen. The cardiac dulness depends entirely upon the presence or absence of hypertrophy or dilatation. It is in general enlarged both to the right and the left, and neither the superficial nor deep dulness undergoes a change in position on alteration of the posture. The heart sounds, when uncomplicated by any lesion, are commonly accentuated, but in cases with a tendency to cardiac failure they may undergo weakening.

Diagnosis.—No one physical sign is really pathognomonic of adherent pericardium, but the diastolic rebound of the parietes, along with immobility of the cardiac dulness in varying postures, is of great value. The systolic retraction of the region around the apex-beat is of some importance, along with other appearances, especially the diastolic collapse of the cervical veins, but these in themselves may occur apart from obliteration of the pericardial sac. The diagnosis of the condition must rest upon the assemblage of the symptoms which are present. If there be a tendency to pulsus paradoxus, diastolic collapse of the cervical veins, systolic retraction of the epigastrium and intercostal spaces to the left of the apex, a fixed position of the apex beat in all postures, immobility of the area of cardiac dulness, diastolic rebound of the chest wall, and inspiratory retraction of the lower end of the sternum, the diagnosis may be regarded as absolutely free from doubt. The character of the heart sounds is too variable to have any importance. A history of previous pericarditis is always of use.

Prognosis.—In simple pericardial adhesions, the outlook cannot be regarded as serious, since the heart is surrounded by lung tissue, marked by such a high degree of extensibility and elasticity as to obviate any serious interference with the cardiac movements. If in its development there has also been pleurisy, the possibility of pleural adhesions has to be remembered, as their existence is a serious feature, and would render the prognosis much graver if they were suspected. If obliteration of the pericardial sac is associated with valvular lesions, the outlook is of much more serious import. The hypertrophy certain to result from the combined internal and external disturbances leads the heart to grow beyond its nutritive possibilities, degeneration ensues, and sudden death from asystole is common.

Treatment.—During the stage of resolution in pericarditis, it may be possible to aid natural processes, and obviate pericardial adhesions. The employment, therefore, at this stage of such remedies as the iodides, along with counter-irritation externally, should be adopted, and if there be any appearance of weakness of the heart, it will certainly be advisable to add one of the cardiac tonics. After the formation of adhesions, treatment can necessarily only be symptomatic. Careful regulation of exercise and rest should be made, so as to lessen all excessive physical stress, by which a strain to the heart and injurious fluctuations of intra-thoracic pressure might occur. Food must be carefully arranged so as to prevent any liability to dilatation of the hollow viscera. If cardiac failure makes its appearance, it must be treated on the general principles which will be laid down in connection with valvular disease.

MEDIASTINAL PERICARDITIS.

It has already been mentioned incidentally, that in pericarditis the morbid process may pass beyond the structures immediately involved, and bring about thickening of the mediastinal structures with various adhesions. Griesinger and Kussmaul brought forward a group of symptoms by means of which they considered it possible to diagnose the disease. The subject, however, is one regarding which a considerable amount of doubt is present; the most important work on it in recent times is that of Harris.

Etiology.—It cannot be denied that the ordinary causes of pericarditis may give rise to such extension of the morbid processes as to involve the tissues of the mediastinum, but some of the cases which have been described by different writers were produced by affections primarily of the mediastinal glands, lungs, or pleuræ.

Morbid anatomy.—Many different forms of pericarditis—fibrinous, serous, purulent, or hæmorrhagic—have been described, along with different secondary affections of the myocardium. Many pulmonary affections have also been found. The ordinary character of mediastinal pericarditis, however, is the presence of membranous or funicular adhesions directly or indirectly uniting the base of the heart and the great blood vessels to the surrounding textures, more particularly those of the mediastinum, sternum, œsophagus, and vertebral column.

Symptoms.—Some of the cases which have been diagnosed as belonging to this group have presented such clinical features as breathlessness, cyanosis, and œdema, but the facts upon which stress has been chiefly laid have been found by physical examination. The pulse has commonly been somewhat empty and of moderate pressure, but the characteristic which has led to most remark has been that a fall of pressure or a tendency to intermission has occurred during inspiration. The pulse has therefore been termed the *pulsus paradoxus*, or *pulsus inspiratione intermittens.* The cervical veins have been observed to swell up during forced inspiration and to subside on expiration. Inspiratory retraction of the epigastrium, and systolic retraction of intercostal spaces surrounding the apex-beat, have also been noticed. A fixed position of the apex-beat in every posture has been noticed. Enlargement of the area of cardiac dulness and different alterations of the sounds of the heart have been noticed, but they are not of much importance.

Diagnosis.—The recognition of such a form of pericardial affection as this, rests upon a somewhat uncertain foundation. The two features which have been relied upon in the determination of the affection are the behaviour of the arterial pulse and the cervical veins. The paradoxical pulse has, however, been observed in several affections of the pericardium other than that under discussion. It has been seen also in aneurysm, and also in persons to all seeming perfectly well. It cannot, therefore, be accepted as infallible evidence of adhesions surrounding the great vessels. The inspiratory distension of the cervical veins must be admitted to have rather more weight than the characters of the pulse, especially when it is associated with other symptoms denoting a pericardial affection.

Prognosis.—The future of any case in which a diagnosis of adhesions about the base of the heart and great vessels, will entirely depend upon the conditions associated with the affection.

Treatment.—The treatment will need to be carried out on the lines already laid down for pericarditis, but each symptom must be met as it arises. There seems to have been a greater tendency towards cardiac failure in those cases which have been diagnosed as mediastinal pericarditis, than in the more usual types of pericardial disease, and the management has therefore more urgently required the administration of diffusible stimulants and cardiac tonics than in the latter.

PNEUMO-PERICARDIUM.

The presence of gas in the pericardial sac, except in small quantities as the result of post-mortem decomposition, constitutes one of the rarest diseases.

Etiology.—The causes of pneumo-pericardium fall naturally into two classes—extension of morbid processes from adjacent structures, and traumatism from the outside. Belonging to the former class are extension from pyo-pneumothorax, pulmonary cavity, cancer of the œsophagus, perforating gastric ulcer, and hepatic abscess producing a communication with the pericardium on the one hand, and the stomach on the other. Openings into the pericardial sac through the chest wall form the great proportion of the causes belonging to the second category, gunshot wounds, incisions, and stabs; but fracture of the ribs has also produced the affection, while an interesting case of perforation of the pericardium by an oriental juggler, on attempting to swallow an instrument, has been described.

Whether gas may be spontaneously developed within the pericardial sac, cannot be regarded as definitely settled. A number of instances have been recorded in which such a causation was accepted. No doubt the pericardial sac on post-mortem examination frequently contains a little gas, but inasmuch as these have been instances in which decomposition with all its possibilities had occurred to a considerable extent, they cannot be regarded as proof of spontaneous generation of gas within the sac. It has further to be remembered that, although the pericardial sac is composed of a tough, fibrous envelope, it is yet possible that a small communication might be established between it and the adjacent pleural cavity. Such must be the explanation of some interesting cases which are on record. Spontaneous pneumothorax appears to take place, and in it, as well as in the analogous pneumo-pericardium, when the small channel communication has healed up, the air is rapidly absorbed. Since the discovery of the *Bacillus aërogenes capsulatus* by Welch, certain of the difficulties connected with the possibility of spontaneous pneumo-pericardium have been to some extent cleared up. The organism produces subcutaneous emphysema in connection with wounds, and is associated with the presence of gas in the blood and the tissues, as well as in the pericardium, pleura, and peritoneum. In this way, when connected with traumatic causes, pneumo-pericardium may take place.

Morbid anatomy.—The presence of gas within the pericardial sac is invariably attended by pericarditis, and there is therefore, in addition to the gas, a certain amount of exudation which is, as a rule, either purulent or hæmorrhagic. The pericardium, when exposed by the removal of the anterior chest wall, is usually bulged forwards in consequence of distension, but this does not occur when a method of exit has been provided by some channel of communication. When the pericardium has been inflated and stretched, the gas has escaped on incision with a hissing sound and a fœtid odour. Both the investing and reflecting membranes present the appearances of pericarditis with a fibrinous exudation, and a certain amount of organisation. A yellowish, brownish, or reddish fluid with fœtid characters has always been observed. Some retraction of the lungs and depression of the diaphragm have almost invariably been seen.

Symptoms.—The affection is accompanied by some of the general symptoms of pericarditis, marked, however, by a greater tendency to rigors, fluctuating temperature, and profuse perspirations. Sleeplessness and delirium are also common; dyspnœa, cyanosis, and œdema are often present. Præcordial pain, often of a severe anginous character, along with palpitation and syncope, are frequently found. The pulse may present many features, but the vessel is somewhat empty and of a low pressure, while the pulsations are irregular and unequal. A bulging of the præcordia sometimes occurs, but much more commonly there is simply disappearance of the visible cardiac pulsation, and the feebleness or absence of the impulse is the most prominent symptom on palpation. On percussion over the præcordia, a remarkable divergence from healthy conditions may be made out. Instead of the ordinary cardiac dulness, there is a tympanitic percussion sound, and this sometimes is accompanied by a cracked-pot sound. It is of importance to bear in mind that the percussion results vary on changing the posture. While recumbent, the tympanitic percussion sound may be present over the entire præcordia, but while sitting upright the lower portion of the præcordia becomes dull from the gravitation downwards of fluid. The normal cardiac sounds are greatly obscured by a remarkable series of accompaniments which are perfectly pathognomonic of the disease. There may be some friction sound, and there may be endocardial murmurs, but these are entirely overshadowed by splashing noises like those of a churn or water-wheel, occurring with the pulsations of the heart, and therefore rhythmic or arhythmic according as the action of the heart is regular or irregular. These different sounds may sometimes be heard at some distance from the patient. The course of the disease is mostly unfavourable, and in the majority of cases death has occurred within a few days, or within a few hours. Cases in which the affection is due to traumatic causes are more favourable than those in which fistulous communication has produced the disease.

Diagnosis.—The recognition of the disease should not present much difficulty. The tympanitic sound on percussion, altering its position according to the posture of the patient, and the wonderful combination of sounds on auscultation, are perfectly pathognomonic. The two difficulties lie in the possibility of mistaking pneumothorax on the one hand, and gastric dilatation on the other, for pneumo-pericardium. With a little care in the analysis of the physical signs of pneumothorax, there should be no dubiety, since the condition of the breath and voice sounds over the region of the lung should furnish clear evidence of the pleural lesion, while the presence of an approximately normal area of cardiac dulness, even if altered in position, will render the matter clear. In gastric dilatation an area of tympanitic percussion sound may be found in the lower portion of the præcordia, and on auscultation there may be splashing sounds accompanying the movements of the heart. In such cases, however, the upper part of the præcordia will never be found to be tympanitic, and on causing the patient to assume a sitting posture the splashing sounds will disappear.

Prognosis.—Pneumo-pericardium is always a serious condition, but the outlook is much graver in instances produced by a fistulous communication with some other viscus, than in those produced by traumatic causes. In the former case the primary lesions are in themselves calculated to produce fatal results, being so often of the nature of septic or malignant changes. In the latter a wound may heal up rapidly, and the air be speedily absorbed.

Treatment.—When the disease is produced by communication with some adjacent lesion, the treatment can only be symptomatic, and for the most part palliative. In traumatic cases, strict antiseptic treatment must be adopted, while the general condition of the circulation is watched so as to obviate tendencies to cardiac failure. If pericarditis is a prominent feature, it must be treated on the principles already stated, and if there be much exudation, more especially if it be purulent in character, it must be removed by appropriate means.

PERICARDIAL TUBERCLE.

Since the observations of Corvisart, tuberculous lesions of the pericardium have been well known. Simple pericarditis may occur in tuberculous subjects without any of the appearances characteristic of tubercle. With this we have at present no concern; but tubercle may occur in the pericardium in its most distinct forms, and may be primary or secondary.

Etiology.—This pericardial affection has been found in every age, from 8 months to almost 90 years. It is more common in males than females. When primary, the disease can only be explained on the assumption, in those who are very young, of a congenital affection, while in older patients it certainly speaks for inherent weakness, or some previous disease of the pericardium, which has rendered the serous membrane more vulnerable. The secondary form of the affection is most usually the result of tuberculous lesions of the pleura, lung, or glands. The latter are more especially a source of infection, and many instances regarded as primary are really produced by secondary implication from glandular affection. As part of an acute general tuberculosis, the affection of the pericardium is rarely, if ever, found without coincident lesions of the other serous membranes.

Morbid anatomy.—Two well-marked groups are formed by the pathological appearances in this disease. There is, first, tuberculous pericarditis characterised by fibrinous and serous exudation. The fibrinous exudate manifests a certain amount of organisation, and in the fibrous tissue so formed tuberculous nodules are found, with giant cell systems and other unmistakable appearances. Sometimes, while no definite tubercles are present, the purulent exudation within the sac contains tubercle bacilli. There is, secondly, the less common type, which may be called pericardial tubercle, in which, with comparatively little exudation, there are distinct tubercles, or even larger tuberculous masses. In all cases there is a considerable tendency towards hæmorrhage into the sac, and superficial ulceration of the serous membrane is not uncommon; it has sometimes been so serious as to involve perforation of the cardiac muscle and rupture of the heart.

Symptoms.—Tuberculous affections of the pericardium are sometimes absolutely latent throughout their whole course, and are only discovered on post-mortem examination. In many cases, however, there are general symptoms of a tuberculous character, such as fluctuating pulse rate and temperature curve, profuse perspirations, rapid emaciation, and progressive debility—the appearances, in short, of hectic. The local evidences require to be sought for, as they are rarely present in such a form as to attract attention. When they are present, the local symptoms and physical signs are simply those of an ordinary case of pericarditis, distinguished, however, by a greater tendency towards chronicity.

Diagnosis.—The recognition of pericardial tubercle in any of the forms mentioned may be effected with comparative ease, if there be a combination of such general features as are commonly attendant upon tuberculosis, along with the unmistakable physical signs of pericarditis, and the diagnosis will be easier if tuberculous lesions are present in any of the neighbouring viscera. In doubtful cases, presenting persistent symptoms of chronic pericarditis without clear evidence of the presence of tubercle, it may be advisable to resort to aspiration in order to determine the nature of the exudation.

Prognosis.—This is always unfavourable in tuberculous affections of the pericardium, since the disease, as a rule, tends towards a fatal result, usually from cachexia. No doubt some cases occur in which, as the result of fibrous changes, a certain amount of recovery ensues. Even in such cases, however, the tuberculous process is apt to follow its usual course in some other part, and a fatal result is therefore likely to ensue.

Treatment.—While an attempt is made to obviate the tuberculous tendency of the disease, every means must be adopted to minimise the local change, and to avert any liability to cardiac failure. The general management, in short, includes such means as are suitable for tuberculosis in general, in addition to such methods as have above been seen to be useful in pericarditis. If there be so much exudation as to interfere with the functions of the heart, it may be necessary to perform aspiration.

PERICARDIAL SYPHILIS.

The invasion of the pericardium by syphilitic is less common than by tuberculous changes, and but few instances are on record.

Etiology and morbid anatomy.—In most of the cases which have been recorded, the affection took place as a late secondary or tertiary manifestation of the disease, but one instance has been seen in a newly born child. The development of characteristic gummata is rare, and much more frequently there is some infiltration leading to the formation of fibrous tissue, resulting in pericardial adhesions, the explanation of which is found in some other organ.

Symptoms and diagnosis.—The affection has remained in most instances unnoticed during life, and the lesions have been discovered on post-mortem examination. The lesions therefore present a contrast to those which are found in the case of invasion of the myocardium. The determination, therefore, of a syphilitic lesion of the pericardium can only be present when, in addition to proof of the existence of syphilis, there are physical signs of pericarditis.

Prognosis and treatment.—If the affection were in any instance diagnosed, the outlook would entirely depend upon the amenability of the disease to appropriate remedies, and it might therefore be regarded as an instance of therapeutic prognosis. In its management, iodide of potassium would necessarily be the most important remedy for internal use, while the application externally of the simpler mercurial ointments might aid in the removal of the lesion.

PERICARDIAL NEOPLASMS.

These affections of the pericardium are uncommon. It will be convenient, along with malignant changes, to include certain parasitic invasions, particularly hydatids and actinomycosis.

Etiology and morbid anatomy.—Primary cancer and sarcoma are extremely rare. One instance only, as a matter of fact, appears to be absolutely established. Secondary developments of these affections are, however, somewhat less infrequent, usually associated with malignant disease of the mediastinum, glands, pleura, lung, or œsophagus. In such cases the characteristic structural features of the disease which is present are found along with infiltration of the serous and subserous tissues.

Hydatid cysts have been recorded in several instances. None have ever been recognised during life, but the lesions referred to were discovered on post-mortem examination. Actinomycosis has been discovered at least twice in connection with the pericardium, associated with characteristic lesions in other organs.

Symptoms and diagnosis.—Malignant invasion of the pericardium gives rise to what may be termed pathological curiosities, seeing that the clinical effects are inconspicuous, and the recognition of the lesion is practically impossible. None of the cases of hydatids were recognised during life, and the symptoms are therefore unknown. The same is true as regards actinomycosis. The diagnosis of this group of affections could only be possible if there were characteristic local appearances of pericardial implication in the presence of the special disease elsewhere.

Prognosis and treatment.—The outlook in all cases belonging to this class is absolutely unfavourable, and the treatment can therefore only be symptomatic.

HYDROPERICARDIUM.

The pericardial sac normally contains a small amount of clear, yellowish fluid, usually from 5 to 10 c.c. in amount, but sometimes amounting to 100 c.c. If this amount is surpassed, the condition must be regarded as pathological, constituting hydropericardium, or pericardial dropsy. This is a serous transudation analogous to hydrothorax and ascites.

Etiology.—Hydropericardium is necessarily a secondary affection, having its origin in the various conditions which produce œdema. It is caused by general venous stasis having its origin directly in valvular disease or myocardial affections, but it may arise indirectly from pulmonary, pleural, or renal disturbances. It may be produced locally by pressure upon the cardiac veins in consequence of some new formation connected with the heart, pericardium, or mediastinum. It may further have its origin in cachetic conditions, acting through the blood. The conception of hydropericardium "*ex vacuo*" cannot now be seriously entertained. There is always a negative pressure in the pericardial sac, no matter how small may be the quantity of fluid which it contains, and it is impossible to conceive of any change in the physics of the thorax sufficient to act so powerfully upon the serous membrane as to induce transudation.

Morbid anatomy.—The fluid found in the pericardial sac is clear, with a yellowish tint and a dichroic tendency. It is occasionally reddish in consequence of the presence of blood, or it may have a dirty brownish hue. Its specific gravity is, as a rule, about 1015. Sometimes there is a flocculent appearance from the presence of endothelial cells, which are usually granular, and which are accompanied in some instances by round cells, also showing a granular tendency. Small masses of fibrin are often present. Sometimes cholesterin crystals have been seen. The amount of the transudate often reaches 1000 c.c., and this has often been greatly exceeded. The sac is distended according to the amount of fluid which is present, and it shows distinct fluctuation. The serous surfaces are smooth, shining, and almost always free from any fibrinous exudation, while they are pale on account of pressure on the vessels of the membrane. The pericardium is often thickened, but sometimes it is abnormally thin. The subepicardial fat is diminished in quantity, and the tissues underlying it are frequently pale and even œdematous. The muscular tissue of the heart is pale and flabby. There may be œdema of the cellular tissues of the mediastinum, and there is often compression of the lower portions of the lungs.

Symptoms.—Hydropericardium produces symptoms which are usually but one part of a great series, and its appearances are commonly overshadowed by the clinical features belonging to the conditions from which it arises. As a rule, the temperature is normal, or even below normal. There are often uncomfortable sensations of oppression in the chest, while dyspnœa, cyanosis, and œdema are, in at least some degree, of constant occurrence. Syncopal tendencies are common, and there may be a soporose inclination, often with a mild muttering delirium towards evening. The attitude is not infrequently that of orthopnœa. The pulse is usually empty, frequent, and irregular. The veins of the neck are turgid, and the præcordia may be prominent. Weakening of the cardiac impulse is often observed, and the vocal fremitus over the præcordia is lessened. There is an increased area of cardiac dulness, which assumes the form that has been seen to be characteristic of pericardial exudation. The heart sounds are feeble, and if murmurs have previously been present, they lose their intensity. It need not be said that a friction sound is absent. The liver is often enlarged, as well as the spleen, and ascites is frequently present. The renal secretion is diminished and often albuminous, while œdema of the bases of the lungs or hydrothorax may be discovered.

Diagnosis and prognosis.—The recognition of hydropericardium must rest upon the presence of signs of effusion, with the absence of any proof of pericarditis, but the diagnosis is facilitated by the existence of such causes as are known to produce it. The outlook in dropsy of the pericardium entirely depends upon the causes which have induced it, and if these are remediable, the prognosis is more hopeful than when they are permanent.

Treatment.—Seeing that hydropericardium is but a symptom of some primary affection, its management depends upon the treatment of the original disease, and in addition to such appropriate methods, the use of dry food along with diuretics and purgatives will be indicated. If the amount of fluid causes much interference with circulatory functions, it will be necessary to have recourse to paracentesis.

HÆMOPERICARDIUM.

As has already been seen, blood may be present in several forms of pericarditis, more especially when occurring in the course of scurvy, cancer, and tubercle, as well as in renal diseases and alcoholic conditions, but hæmopericardium is to be regarded as a different affection from these.

Etiology.—The causes of hæmopericardium may be traumatic or idiopathic. Amongst the former class are wounds inflicted by projectiles or sharp instruments, but the affection occasionally occurs in consequence of a blow or a fall. In these latter cases, however, there has probably been some structural alteration predisposing to hæmopericardium. The pathological factors producing hæmopericardium are rupture of the heart, of a coronary artery, of the aorta, or of an aneurysm; but ulceration of the heart or of one of the blood vessels is sometimes to be made out.

Morbid anatomy.—The amount of blood contained in the pericardium varies considerably, and depends entirely upon the cause of the escape and the time which has been occupied. The blood may be entirely coagulated, or altogether fluid. Most commonly, however, a certain amount of coagulation has taken place, although some of the blood usually remains fluid. Careful examination reveals the source of the escape.

Symptoms.—The clinical features presented by the affection are somewhat variable. Sometimes the events unfold themselves with a swiftness that is dramatic; at other times there is a protracted and lingering passage into a condition of asystole. Occasionally a sharp stabbing pain precedes the incidence of the affection. Giddiness and faintness are often the first symptoms of disaster; but no matter how the onset occurs, there is a quicker or slower passage into unconsciousness. The aspect is that of deathly pallor, usually with a cold perspiration bathing the features. The pulse is empty, feeble, and irregular. On examining the chest, there is usually a total absence of all characteristic appearances, except in such cases as have developed slowly, when the area of dulness may be somewhat enlarged.

Diagnosis.—The recognition of the nature of the event is only possible when the presence of some affection likely to produce it has been previously established. If such a condition is known to have been present, and is followed by sudden collapse, the condition may be inferred.

Prognosis and treatment.—With very few exceptions, the prognosis is absolutely hopeless, and no methods of treatment are of any avail.

G. A. GIBSON.

DISEASES OF THE ENDOCARDIUM.

The endocardial membrane is liable to a series of affections, most of which are secondary to disease elsewhere. However the various lesions of the endocardium may terminate, they practically fall into two groups—endocarditic and degenerative. Of these two classes, the former will be dealt with in the following section; the degenerative lesions will be discussed in that which succeeds it on the Chronic Affections of the Valves.

ENDOCARDITIS.

The classification of endocarditis, which has, at least in this country, been mostly adopted, is unsatisfactory. It has been customary to divide the disease into an acute and a chronic form, and the acute has been further subdivided into malignant and simple varieties. It is impossible to regard this arrangement as adequate. Every form of acute endocarditis is attended by, if not due to, the presence of micro-organisms, and the same pathogenic agents have been found in those forms of disease commonly looked upon as malignant, and in those which are not. The lesions also found after death are often similar in cases which would be diagnosed as belonging to different classes. From the clinical point of view, further, the distinction between malignant and simple acute endocarditis is attended by great difficulty. Many cases which appear for a time to be free from any malignant tendency undergo a disastrous change in their clinical appearances, while others, commencing apparently with serious symptoms, take a favourable turn and terminate satisfactorily. It therefore follows that a different classification is advisable; in the following pages, accordingly, the disease will be grouped under the forms of acute, subacute, and chronic endocarditis.

The frequency of endocarditis cannot be determined with such accuracy as in the case of pericarditis, seeing that the symptoms relied upon for diagnosis are far from uniform, but, judging by Gillespie's statistics of the Royal Infirmary of Edinburgh, the disease is by no means so common as pericarditis. In a total of 2368 cases of heart disease, admitted between 1891 and 1896, there were only fifty diagnosed as endocarditis, twenty-six males and twenty-four females, of whom fourteen and eighteen respectively died. There can be no doubt that this somewhat understates the frequency of the disease, which must have been a frequent attendant upon many other diseases under treatment.

Etiology.—Among **predisposing causes** of endocarditis, some place must be allowed to the influence of heredity. All who have worked at the subject are at one in regarding inherited liability to endocardial changes as an important factor. With regard to sex, it has often been stated that simpler forms of endocarditis are more frequent amongst men, while graver types are most commonly found in women; but the recent observations of Kelynack, and of Kanthack and Tickell, throw doubt upon the correctness of this view, seeing that more than 60 per cent. of their fatal cases of endocarditis belong to the male sex. As to age, the affection is essentially a disease of the active period of adult life, its incidence being most frequent between the ages of 20 and 40. Previous disease of the valves is a most important agent predisposing to endocarditis, a fact which was first observed by Paget. Amongst predisposing agencies must finally be mentioned a large number of previous diseases, some general, some local, but as they are frequently associated in a more direct etiological connection, it is more satisfactory to place such affections among the exciting causes of endocarditis.

Exciting causes.—The exciting causes of endocarditis are almost entirely connected with infective processes and constitutional affections. Scarlet fever, measles, smallpox, influenza, erysipelas, pneumonia, and septicæmia furnish a considerable proportion of cases. Enteric fever, diphtheria, malaria, and tubercle are only found, in exceptional instances, to underlie endocarditis. The rheumatic group of diseases comprises the most frequent causes of the affection; indeed, they give rise to the largest proportion of all cases of endocarditis. The researches of Poynton and Paine have gone far to prove the microbic origin of acute rheumatism and its cardiac effects. Numerous statistics have been compiled with reference to the frequency of endocardial complications in acute rheumatism. The figures which have been published vary enormously, and it would serve no good purpose to quote them; seeing that they have been gathered from many climates, and from patients of very different ages, some discrepancies might be expected, but the excessive divergencies are probably due, at least in part, to the influence of the personal equation. There is one observation which must be added—acute rheumatism during childhood produces more serious effects on the endocardium than is the case in adult life. Many cases of endocarditis are due to what may be called latent rheumatic affections, chief amongst which is chorea; but in addition to chorea, other manifestations of a rheumatic taint may throw an etiological light upon endocarditis. Erythema nodosum has been frequently found in this connection, and rheumatic nodules tell the same tale. The group of disorders usually termed gouty, is occasionally the starting point for endocarditis. Nephritis not infrequently precedes attacks of acute endocarditis. In some of these cases the same morbific agent may be the cause both of the renal and cardiac lesions, but this can scarcely be the case in regard to chronic kidney disease, where some secondary infection is probably found.

It is more than probable that bacterial infection is present in every case of acute and subacute endocarditis. Micro-organisms have been discovered in every form of acute endocardial vegetation. Pyogenic microbes are most frequently seen, but many bacilli and cocci have been detected. Certain special forms have been described, such as the *Bacillus endocarditidis capsulatus griscus* of Weichselbaum and Netter; the *B. immobilis et fœtidus* of Fränkel and Sänger; and the *Micrococcus endocarditidis rugatus* of Weichselbaum. In most instances only one organism has been found, but cases have been described in which several varieties have coexisted. When embolism occurs in acute rheumatism, the same organism is found in the primary and secondary lesions. Many investigations have been carried out with the view of determining how such organisms produce endocarditis. It has been held, on the one hand, that the mere presence of organisms in the blood was a sufficient cause of endocarditis; while it has been asserted, on the other, that such organisms are carried by the blood into the substance of the endocardium, and produce embolism there. The question is surrounded by difficulties arising from the fact that the arterial valves of the heart are destitute of blood vessels. All observers are agreed that experimental endocarditis can be most easily produced by the introduction of organisms after the

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endocardium has been wounded, as for example by the introduction of a stylet through one of the arteries or veins. Such mechanical injuries are not, however, necessary, since the introduction of streptococci or staphylococci is in itself sufficient to set up endocarditis. By cultivation, organisms have been obtained from the vegetations of endocarditis destitute of malignant or ulcerative features, which when injected into the rabbit gave rise to most extensive lesions. It is probable that the blood may sometimes contain a poison of sufficient virulence to produce endocarditis without previous lesions; while in other instances, previous lesions have a determining influence. This is not only a fact derived from the results of experiment, but is an observation of everyday occurrence in morbid anatomy. It seems probable, from the recent observations of Sidney Martin, that certain of the chemical substances produced by the action of staphylococci have a destructive influence upon the structure of the heart.

In most of the diseases which have been referred to as factors in the production of endocarditis, it seems probable that double infection is at work. As is well known, whatever may be the special cause of scarlet fever, most of its serious symptoms in grave cases are due to the presence of streptococci or staphylococci. So, in the case of the diseases which have been mentioned, it seems probable that some pathogenic organisms have obtained entrance into the blood, and have added their effects to the influence of the primary affection. To all these causes of endocarditis must be added the influence of long-continued muscular strain. This factor, especially when associated with pathogenic conditions of the blood, produces disastrous effects upon the lining membrane of the heart.

Morbid anatomy.—Acute endocarditis is much more frequently found on the left than on the right side of the heart. It is believed that two factors lead to this disproportion—the higher blood pressure within the cavities of the left side, and the arterialised condition of the blood which they contain. This statement must be taken with the reservation that amongst those varieties clinically termed malignant, a somewhat larger proportion affect the right side, the explanation of which seems to be that septic poisons are more likely to encounter the right side of the heart on entering the circulation.

Any part of the endocardium may be affected, but certain portions are specially liable to implication. The disease occasionally attacks the membrane lining the cavities of the heart, when it is termed parietal; but in the overwhelming proportion of cases it only attacks the valves, when it is called valvular endocarditis. The aspect of the valves almost always affected is the surface exposed to the blood stream—the auricular surface of the venous, and the ventricular surface of the arterial valves. The lesions are not situated at the edges of the cusps, but at a short distance from them, at that part which comes into most forcible contact with the opposing cusp.

A long series of lesions is found in endocarditis, obviously dependent upon three conditions—the severity of the process, the extent of the reaction, and the duration of the disease. The lesions cannot be definitely separated from one another, and they form a chain, the widely dissimilar ends of which are united by a perfect series of links.

Acute endocarditis.—The first stage in the production of acute endocarditis is some dulness of the membrane from the products of irritation, with cloudy swelling of the tissues. By the increase of these changes, a thickening of the part occurs, giving rise to some prominence, which may be white, grey, or green in tint. The elevation can usually be removed by gentle rubbing, and it leaves a distinct abrasion of the surface. Shortly afterwards, smaller projections take their origin from the surface and edges of the little prominence, and give rise to a cauliflower-like excrescence. The further pathological progress may diverge at this stage, the changes may become less acute, in which case the mass assumes a firmer consistence; or, on the other hand, the thickened portion may give way and produce an ulcer with the liberation of the vegetations. This necrotic process may be so severe as to produce perforation of a cusp, or of the wall of the heart itself.

The microscopic examination of acute endocarditis shows at first the presence of small round cells caused by the proliferation of the connective tissue corpuscles. They occupy the fibrous layer beneath the endothelium, and extend into the areolar tissue next to the myocardium, and even invade the intermuscular septa. The endothelium covering the affected part breaks down, so that the small cell infiltration projects freely, and deposits of fibrin take place upon the surface. In the spaces between the fibrinous layers, as well as upon the free surface, are masses of organisms, most usually staphylococci, streptococci, and diplococci. Many other forms of organism may be found.

During the development of these changes, there is a tendency towards the separation of fragments which are carried as emboli along with the blood stream, and give rise to simple infarction, or pyæmic abscess, according to circumstances. In endocarditis of the left side of the heart, the organs which suffer are—in order of frequency—kidney, spleen, brain, and liver. When the affection involves the right side of the heart, the embolic processes are usually confined to the lungs.

Subacute endocarditis.—In this form of endocarditis the processes are much more gradual, whether on account of the less severity of the process, the greater extent of the reaction, or the long duration of the disease. On account of the greater tendency to the formation of fibrous tissue, the resulting lesions are more vegetative or verrucose in their type; the granulations are more permanent; there are more adhesions between the cusps; and there is less loss of tissue.

The appearances presented by the diseased structures are—thickening of the endocardium, adhesions of the cusps to one another, and firm vegetations. Microscopic examination shows an infiltration of the endocardium with small round cells, and the granulations are formed of firm fibrous tissue, upon the surface of which recent deposits of fibrin are commonly observed. Micro-organisms are often determined in such cases, and they are usually identical with those which are found in the acute variety of the disease. In a proportion of cases no organisms can be detected. They have died before the stage which is present has been reached.

Chronic endocarditis.—In this form of the disease, the conservative and reparative processes are still more marked; and it is therefore characterised by the presence of firm vegetations, resistent thickenings, and rigid union of the cusps. A deposition of lime salts very often occurs. The lesions of chronic endocarditis constitute one large and important group of the chronic valvular affections, which are separately described. The appearances in chronic endocarditis are those of deformity of the valves, with or without vegetations; and these lesions produce the various effects of obstruction or incompetence. The microscopic examination shows dense fibrous tissue, somewhat stratified in arrangement, frequently associated with deposits of fibrin upon the surface, in the meshes of which various organisms are sometimes found. In the majority of cases observed there are no organisms, as, even if they have been present at one period, they have ceased to exist. In this, as in the other forms of endocarditis, embolism may occur.

Symptoms.—The clinical features of endocarditis form a somewhat indefinite group, but it is possible to classify most cases under one or other of the three great divisions already mentioned—(1) the acute, malignant, or ulcerative; (2) the subacute, verrucose, or vegetative; (3) the chronic, sclerotic, or contracting. It is, however, often difficult to place certain instances of the disease under any one of these divisions, inasmuch as there may be doubt, on the one hand, whether they should be placed in the first or second group, and, on the other, whether they should be relegated to the second or the third.

Acute endocarditis.—Two main tendencies are manifested by cases belonging to this division—the typhoid and the intermittent.

Typhoid type.—In this form, the temperature is usually high and sustained, the pulse frequent and feeble, the respiration shallow and hurried. There are profuse perspirations; the tongue is, as a rule, dry, often glazed and fissured; but in the earlier period of the disease there may be a central fur bordered by red edges. Meteorism and diarrhœa are common,and melæna occasionally occurs. The spleen may be enlarged; albuminuria is very frequent, and hæmaturia is sometimes present. Rosy papules appear upon the skin, and sometimes become purpuric, while the mucous membrane of the mouth and conjunctiva may have extravasations. These appearances are produced by emboli, and the same organisms have been found in them as on the endocardium. An apathetic or soporose condition tends towards coma in some instances, and towards delirium in others. The results of physical examination are often indefinite. The pulse is empty, compressible, frequent, irregular, and shows dicrotism, or even hyperdicrotism. The cardiac impulse is weak, and the sounds feeble. If the affection does not cause death at an early period, the area of cardiac dulness may enlarge, while the waning sounds become muffled or impure. Systolic murmurs in the mitral and tricuspid areas are usually present; but presystolic or diastolic murmurs may occur in those regions, while systolic and even diastolic murmurs may be developed at the base.

The course of this typhoid form is usually rapid, and cases have proved fatal on the second day of the disease. Death usually occurs from acute general infection, or from heart failure in consequence of extension of the process to the myocardium. Embolic implication of the central nervous system or pulmonary complications may, however, be the cause of death. Less frequently the disease passes into a subacute form, and may even, although rarely, become chronic.

The *intermittent type* of acute endocarditis is distinguished by septicæmic features. The temperature is irregular and intermittent; the pulse rate fluctuates widely; and the respirations are very variable in frequency. Profuse perspirations occur at the termination of the febrile periods. Complete anorexia with furred tongue and diarrhœa may be considered the rule. The spleen is almost always enlarged. The serous membranes are often affected, while hæmaturia or hemiplegia may indicate embolic processes in the kidney or brain. Cerebral symptoms, such as delirium or coma, may take place. Careful examination of the circulatory organs may fail to give any definite information. The pulse is usually empty and irregular, while tending towards dicrotism. The cardiac impulse is feeble, and the præcordial dulness is enlarged. Systolic mitral and tricuspid murmurs, and accentuation of the second pulmonary sound, are almost always present; but as they may occur in any condition of pyrexia, they are not of much value. If obstructive murmurs develop, they yield valuable evidence. Many instances of the disease run a latent course, and are only revealed by some disturbance of the circulatory system, such as the sudden development of a previously absent valvular disease, the abrupt onset of cardiac failure, or the occurrence of embolic processes in other organs. The disease often runs a prolonged course. It may continue in an acute form for weeks, and cause death by asthenia, or by one of its many complications; or it may pass into a less acute form, with occasional exacerbations lasting for months. Sometimes, but rarely, it assumes some still more chronic form. Until quite recently, the end has been almost invariably fatal.

Subacute endocarditis.—This form of endocarditis is frequently unattended by any general symptoms. When occurring as a complication or sequel of some general disease, such as acute rheumatism, it sometimes does not even cause any alteration of the temperature, already elevated, and but little change in the pulse or respiration. Even when taking origin in some chronic disease,—for example, nephritis,—it can sometimes only be detected by careful physical examination of the heart, and this even may be fallacious. On the other hand, the onset of endocarditis may be attended by rise of temperature, acceleration of pulse, and frequency of breathing. There are occasional subjective sensations connected with the heart, such as pain and palpitation, or faintness and giddiness. The pulse becomes empty and compressible, as well as increased in frequency and irregular in rhythm. The cardiac impulse becomes feeble, but in consequence of its excitement the short sharp tap may be mistaken for an exaggerated impulse. The area of dulness is often increased. The results of auscultation furnish many possibilities of error. In every febrile condition the heart is apt to undergo dilatation, leading not only to muffling of the cardiac sounds, but to the development of systolic murmurs from incompetence at the auriculo-ventricular orifices, produced by relaxation of the tissues. Such murmurs furnish no indication of endocarditis, and the only physical signs pathognomonic of endocarditis are obstructive murmurs, such as systolic aortic or presystolic mitral murmurs. When a systolic mitral murmur, however, is developed at a very early period of the febrile attack, it may with justice be regarded as presumptive evidence of endocarditis, seeing that it has occurred before there has been time for the development of regurgitation by means of cardiac relaxation. This has been clearly shown by Sansom. Accentuation of the pulmonary second sound is extremely common. Breathlessness, cough, and other pulmonary symptoms may arise in consequence of implication of the lungs by cardiac weakness. Albuminuria and hæmaturia may ensue as secondary effects of cardiac failure. Headache, restlessness, and sleeplessness may be produced by failure of blood supply to the brain. Embolic processes frequently occur, giving rise to characteristic local effects. Sudden pain in the loins, rigors, and hæmaturia are evidences of renal embolism. Pain over the left hypochondrium, sometimes with shivering, and succeeded by enlargement of the area of splenic dulness, may denote embolism of the spleen. The sudden onset of paralytic effects will prove embolism of the brain. Internal pain, with hæmorrhage from a mucous membrane, may indicate embolism of some part of the alimentary tract; and local absence of the pulse, with a chilly surface, darting pains, and paresis may follow embolism of the artery of a limb. Sudden death from cardiac failure sometimes results from coronary embolism. Erythema nodosum in subacute endocarditis has been attributed to embolism, but as it is always associated with rheumatism, it is more probable that it results from the local action of the as yet unknown poison of that disease. Such complications as pericarditis and pleurisy are rather frequent, more usually from the widespread influence of the primary affection, than by extension. It is probable that myocarditis arises in many instances by direct extension from the diseased endocardium, and that it is the cause of the weak and irregular pulse, the feeble and flickering impulse, and the breathlessness and faintness so often found. Pneumonia is more commonly a precursor than a complication of endocarditis; but secondary hypostatic changes in the lung are extremely common as sequels of endocarditis.

The course of subacute endocarditis is more lengthy than even that of the intermittent type of the acute form of the disease, and it commonly terminates in the gradual recovery of the patient as regards general health, with the development of a chronic valvular lesion as the local result. It must always remain in doubt whether absolute recovery ever ensues.

Chronic endocarditis.—This form of the disease is not attended by any general symptoms, except such as result from the circulatory disturbances to which it leads. Pyrexia is only present if an acute attack has been lighted up, or if some complication occurs. The local symptoms and physical signs of chronic endocarditis are entirely the result of the changes in the valvular and muscular conditions to which it leads, and they fall more naturally under the group of chronic valvular lesions, to which the reader is now referred.

Diagnosis—Acute endocarditis.—The recognition of acute endocarditis is sometimes a matter of considerable difficulty, as must be apparent from the statements which have already been made in considering the symptoms of the disease. It is scarcely necessary to reiterate what has been said in regard to the physical signs produced by acute endocarditis, but it is necessary to point out that the affection may be mistaken for septicæmia, acute tuberculosis, and enteric fever. Septicæmia is the disease, above all others, for which the affection may be mistaken, and this cannot be matter for surprise, since a large proportion of the cases of acute endocarditis are septic in origin. It therefore comes to be a question, in any given case, whether the septic infection is attended by endocardial changes or not. In both instances there are characteristic features as regards the pulse, the skin, and the temperature, and the sole distinction between cases with and without endocarditis must arise from the physical examination of the heart. Acute tuberculosis is less likely to be a cause of error, since the periodic fluctuations of pulse and temperature and the excessive nocturnal perspirations are more regular in their occurrence, while the wasting is much more rapid. There are, moreover, often definite clinical features of local lesions, for example, of the lungs, of the abdomen, or of the brain, and in almost all cases the heart gives absolutely negative evidence when examined. Enteric fever may present such aberrant symptoms as to lead to difficulty. Every clinical feature of diagnostic value may be absent, and thereby lead to doubt. Even in such cases, nevertheless, the regularity and infrequency of the pulse, and the peculiar characters of the temperature, may be of use in distinguishing the affection from endocarditis, even when cardiac symptoms and sounds are present. It need hardly be added that at the present time any doubtful case would be submitted to bacteriological investigation, and that the blood would be tested for the typhoid reaction.

Subacute endocarditis.—The recognition of subacute endocarditis is not attended by so much difficulty, and it only requires care on the part of the observer during the course of such diseases as are liable to the complication. As in the case of pericarditis, the onset of endocarditis must be diligently looked for during the course of acute rheumatism, acute pneumonia, and every other disease in which endocardial affections are likely to be developed. The only difficulty in the diagnosis of subacute endocarditis lies in the possibility of mistaking simple dilatation from pyrexia for endocarditis. Upon this subject it is unnecessary to dwell further.

Chronic endocarditis.—The diagnosis of chronic endocarditis is simply that of valvular disease, and the only difficulty is to determine whether the affections of the cusps are the result of endocarditis, degeneration, or injury. The subject, however, belongs naturally to the section upon Valvular Disease.

Prognosis.—The forecast of the future in endocarditis depends upon a due estimation of many factors. The character of the primary affection upon which the lesion depends is of the greatest importance, and in most instances where the disease is part of a general infection, almost all depends upon its severity. In many cases of septicæmia, for instance, the cardiac complication is simply an episode; but it must, however, be kept in view that, as the result of mixed infection, fatal endocarditis may arise in the course of an affection of no great gravity. The outlook may be regarded as much more serious in the acute than in the subacute form of the disease, while chronic endocarditis, regarded from the point of view of practical medicine, is simply one aspect of chronic valvular disease, and does not really fall to be considered in this place. The prognosis in acute endocarditis is still serious, but with the light thrown on the disease by modern research, and the help afforded by recent investigations, there is reason for expecting that the affection will, before long, lose much of its former hopelessness. Subacute endocarditis may be regarded generally as tending towards a favourable termination as regards life; it is, nevertheless, sometimes directly fatal by passing into a more serious condition, or leading to certain complications, but its usual termination is seen in the evolution of chronic valvular disease. The prognosis of chronic endocarditis will be mentioned under the Chronic Valvular Affections.

Treatment.—Abundant opportunities are afforded by endocarditis for the employment of therapeutic measures. In acute endocarditis, until quite recently, the only available methods have been found in the employment of antiseptic remedies. Within the last few years our knowledge of serum therapeutics has given the physician a more direct means of meeting the disease. As yet the whole subject is in an experimental stage, but the results are certainly encouraging. As the usual agents in acute endocarditis are pyogenic organisms, the particular form of serum will necessarily be that which is antagonistic to the organism which is present. In some cases bacteriological examination will reveal the organism which is present, and if this be a form of streptococcus, the antistreptococcic serum should be employed. The first dose of this should not exceed 5 c.c., but the quantity may be rapidly increased to 20 c.c. One injection should be given daily with rigid aseptic precautions. If the disease depends upon the presence of the staphylococcus, there is at present no means of combating it by serum therapeutics, but as regards the pneumococcus some recent investigations lead to the hope that its appropriate serum will be found of real utility. These remedies may be employed both as prophylactic and remedial agents, and it need hardly be added that the employment of cardiac tonics and stimulant remedies must be also kept in view.

As regards subacute endocarditis, there will always be an important question as regards prophylaxis. There can be no doubt that in the diseases liable to this complication it is a duty to adopt certain measures with prevention in view. The appropriate treatment for any acute process that may be present must be diligently persevered with, and as an overwhelming proportion of cases arises in the course of acute rheumatism, the use of some member of the salicyl series of drugs will doubtless be followed. The addition of iodide of potassium to the special remedies in use, and systematic counter-irritation over the præcordia, are certainly of utility. As regards other general diseases, specific measures must also be adopted. In the case of gout, for example, colchicum and alkalies should be freely employed, and similarly in all other constitutional or specific affections. Further, as many general diseases are complicated by mixed infection, an effort should be made to detect any microbes which may be present, with a view to antitoxic or antimicrobic inoculation. When subacute endocarditis has been absolutely detected, the lines upon which the primary disease has been treated must still be carried out, while certain additional measures will be required. The necessity for absolute rest, adequate sleep, abundant air, and appropriate clothing requires no comment. Milk diet administered at frequent intervals, especially with the addition of some alkalies, is advisable. In many cases no further treatment is required, except the employment of some counter-irritation, which is best carried out by a succession of small fly-blisters. The application of cold is of doubtful advantage, since it is apt to prove depressant, and the use of hot fomentations or poultices almost invariably proves more useful. In some cases, when præcordial pain is prominent, the employment of leeches is useful, and there never need be any hesitation in adopting the use of morphine. Throughout the course of the disease, cardiac failure must be watched for, as it may ensue from paralytic myocarditis with alarming rapidity. Free stimulation and the use of the cardiac tonics should be adopted if there be any waning of the heart sounds, or arterial pressure. In many cases the use of strychnine hypodermically, along with the inhalation of oxygen, will prove salutary. After the more acute stage has passed off, the diet may be modified, and the remedies mentioned diminished. Absolute rest must for a considerable length of time be insisted on. Iron, arsenic, quinine, and strychnine find an appropriate sphere of action, while the use of iodide of potassium for a long period is eminently advisable.

The treatment of chronic endocarditis will be considered in the next section.

CHRONIC VALVULAR DISEASE.

Disturbances of the functions of the orifices and valves are produced by many different causes, which may be organic or functional in their nature. Although there must, in every instance, even in evanescent valvular affections, be some structural change in the mechanism, yet it is common to regard certain disturbances, in which the valves are themselves healthy, as functional; this conventional phraseology may be retained with the proviso just stated. It may be said that all cases of obstruction have their origin in structural alterations, since the only possible exception to this statement lies in the occurrence of a relative or apparent obstruction at an arterial orifice, produced by dilatation of the channel beyond. Such an *apparent* obstruction, although furnishing at times physical signs closely resembling those found in *absolute* obstruction, cannot be considered as falling under the same head. It is far otherwise with regard to incompetence of the valves, since regurgitation may be allowed by a series of alterations in their mechanism, some of which are almost entirely functional. An arterial orifice, for example, may be simply dilated from long-continued stress; while in the case of the auriculo-ventricular orifices incompetence may be similarly produced by widening of the orifices, by alterations in the papillary muscles or tendinous cords, or by structural lesions in the cusps themselves.

The relative proportion of cases of chronic valvular disease varies considerably in different statistics. This cannot be wondered at, since there are not only such variations as may be expected in different localities, and at different periods, but the method of registration also accounts for certain differences. One physician may classify instances, for example, of aortic obstruction and incompetence under one or other head, according to the principal symptoms, while another might place it under the heading of combined lesions. During the five years 1893–1898, the statistics of the Royal Infirmary of Edinburgh, collected by Lockhart Gillespie, show that 2368 cases of cardiac disease were admitted, of which 1914 suffered from valvular lesions. The facts are as follow:—

Lesions.	Aortic Valve.	Mitral Valve.	Two or More Valves.	Tricuspid and Pulmonary.	Total.
Males . .	354 or 28·5 per cent.	597 or 48·1 per cent.	278 or 22·4 per cent.	12 or 0·96 per cent.	1241
Females .	57 or 8·4 per cent.	523 or 77·7 per cent.	90 or 13·3 per cent.	3 or 0·5 per cent.	673
Total . .	411 or 21·47 per cent.	1120 or 58·5 per cent.	368 or 19·2 per cent.	15 or 0·78 per cent.	1914

The facts embodied in the foregoing table agree in the main with the current beliefs in regard to the occurrence of such lesions. The number of male cases is almost twice that of female cases; but out of these, while aortic disease affected 28·5 per cent. of the males and only 8·4 per cent. of the females, mitral disease attacked 77·7 per cent. of the females, and only 48·1 per cent. of the males. While aortic disease, therefore, accounted for less than one-fourth of the whole, mitral disease was present in more than one-half of all the cases. Affections of more than one valve were recorded in 19·2 per cent. of all the cases, and lesions of the right side of the heart give only a fractional result. In regard to this last point, it is only right to state that numerous cases of tricuspid incompetence must have been under treatment without being recorded, since it is by far the most frequent of all valvular affections.

Etiology.—Chronic affections of the orifices and valves are sometimes the result of embryonic causes, but as congenital lesions of the heart are discussed in a separate section, there fall to be considered in this place only such causes as are operative after birth.

Predisposing causes.—Among predisposing causes, heredity must be allowed to have some influence in the production of valvular disease, as was indeed observed by Lancisi; and to any one who is much concerned in the study of cardiac disease, the frequency of the inheritance of a tendency must be well known. Age is a factor of considerable importance. Early life furnishes the largest proportion of mitral diseases, while middle life is the period when aortic lesions are commonly developed. Sex also plays an important part, since the largest proportion of aortic cases is found amongst men, while mitral disease is more common in women. This is amply borne out by Gillespie's statistics from the Edinburgh Royal Infirmary. Occupation has, since the time of Boerhaave, been recognised as an important factor in the etiology of chronic valvular affections. Within recent times, many authors, of whom Clifford Allbutt is the most important, have shown that long-continued physical stress not only leads to relative insufficiency of the great venous valves through ventricular dilatation, but also to degenerations of the endocardium, more particularly of the left side of the heart, and especially of the aortic orifice and its cusps.

Exciting causes.—Endocarditis, whether in its subacute or chronic form, is the greatest cause of valvular disease, but it produces different results as regards the various valves. As the commonest factor in the production of endocarditis is rheumatism, the importance of this disease is very great, but it operates differently on the various orifices and valves. My Birmingham statistics are sufficient to prove this. Amongst aortic cases, 50 per cent. were associated with personal or family rheumatism, and 50 per cent. were independent of such a connection; while of the mitral cases, 64·4 per cent. occurred in patients who were themselves rheumatic, or had rheumatism in their families, and 35·6 per cent. were free from such a taint. As regards the great venous valves, which are abundantly supplied with blood vessels, it is possible that vascular rupture and hæmorrhage may be the starting-point of chronic valvular disease. Facts in support of this conception have been reported by Garel and myself. Degenerative changes taking place as part of the retrograde processes of advancing years, are fertile sources of valvular disturbances, and it is probable that such degenerative changes are mainly conditioned by two factors—the presence of toxic substances in the blood, whether chemical or organic, and long-continued stress. Valvular diseases owe their origin occasionally to direct or indirect violence, as has been well known since the publication of Corvisart's epoch-making work. The cause of such injuries may be by spontaneous rupture during excessive muscular exertion, or traumatism in consequence of external agencies. Barié has collected thirty-eight cases, in 67 per cent. of which the valvular lesion was due to spontaneous rupture, and in 33 per cent. it was produced by external violence; in 50 per cent. of the total number of cases the aortic cusps were the seat of the lesion, in 42 per cent. the mitral cusps were affected, and in 8 the tricuspid valves were ruptured.

Valvular derangements in which the cusps themselves may be perfectly healthy, have their origin in causes operating entirely upon the orifices, the walls, or the papillary muscles. Severe stress from physical exertion may cause sudden or gradual strain of these structures. Both acute and chronic disease may give weakness and loss of elasticity in them, and structural alterations of the muscular walls, whether myocarditic or degenerative, leads to dilatation. In all these ways a relative incompetence may be brought about without direct impairment of the valves.

Morbid anatomy.—The appearances in chronic valvular disease form a widespread group, the extremes of which are very distant, but form a perfect series by means of connecting links.

Primary lesions.—Endocarditic changes are seen in vegetations or granulations which have become dense and firm from the growth of fibrous tissue. Along with these changes there are adhesions between neighbouring cusps, or even a complete fusion of the adjacent edges. There is also considerable shrinking of the cusps, and occasionally small aneurysms or perforations take place. These processes usually result in obstruction of the orifices as well as incompetence of the valves. Degenerative lesions of the endocardium are common after middle life, and the most common form is a sclerotic thickening of the membrane, often associated with fatty or calcareous changes. Such lesions give rise to shrinking and deformity of the cusps, resulting in obstruction as well as incompetence. New formations are rare sources of valvular disturbance. They occasionally have their origin in the cusps, but are more common upon the walls of the heart. Cases are on record in which valvular disturbances have been produced by such neoplasms, the most famous being that described by Gairdner. Traumatic lesions give rise, as a rule, to obstruction as well as incompetence, since the valvular cusp or tendinous cord that has been ruptured is at once an obstacle to the onward flow and a source of escape. Incompetence of the cusps may take origin in any of the lesions just referred to, but in addition, it may also be produced by widening of the orifice from any of the causes already mentioned, or dilatation of the cavities producing a want of the proper adjustment of the valvular mechanism.

Secondary effects.—Many results are produced by valvular lesions, some being the direct consequences, and others the indirect effects.

As the processes affecting the orifices and cusps are chronic, there is by no means the same tendency to direct extension of the lesion as is the case in the acute and subacute forms of endocarditis. There are, however, a few direct effects upon the tissues, as shown, for example, by the morbid changes set up by friction. Hodgkin long ago pointed out that it is not uncommon to find indurated vegetations produce thickenings and roughness at the points of impact on the interior of the heart and great vessels. The characteristic results of valvular lesions are to be found as the result of disturbance of the blood flow. As regards the heart itself, certain structural alterations follow such disturbance. The first result of any retardation of the blood current is a stagnation leading to distension and the presence of a residuum of blood. The accumulation in the cardiac chambers leads to some dilatation—the retro-dilatation of Adams and Forget—and when the cardiac tissues are healthy and the blood is in a good condition, the heart is led to contract more powerfully. In consequence of such increased activity, hypertrophy is the result. It may be laid down as a general principle that there are some differences between the effects of obstruction and incompetence, but the results of Hamilton's careful investigations bring out some facts at once unexpected and inexplicable. In the case of incompetence there is not necessarily any reduction in the total amount of blood sent out into the general circulation, but there is, as a rule, a diminution of the mean pressure; in consequence of the regurgitation, there is in any chamber behind the faulty valve some dilatation with hypertrophy, and the dilatation is much greater than in the case of simple obstruction. As regards obstruction, there is greater likelihood of diminished outflow into the general circulation with diminished blood pressure and lowering of the general nutrition, while in chambers situated behind the obstruction there is a considerable amount of hypertrophy, with less dilatation than in the case of obstruction. The interesting observations of Samways on the condition of the auricles and mitral disease, may be cited in proof. When obstruction and incompetence are combined, there is at once considerable dilatation and hypertrophy.

The results of valvular lesions upon the different subdivisions of the circulation produce varied morbid appearances. In aortic lesions the chief influence is exerted upon the left ventricle, which undergoes a variable amount of dilatation and hypertrophy; comparatively little interference with the other chambers of the heart occurs so long as the mitral cusps are competent and the ventricular walls undergo no degenerative changes. In mitral lesions the left auricle is directly implicated, and from its relative weakness backward effects are apt to tell through the pulmonary circulation upon the right ventricle, which undergoes some dilatation and hypertrophy. When the tricuspid valve gives way through dilatation of its orifice, the right auricle also suffers, and there is no further means of averting disturbance of the general venous system.

In the case of mitral lesions there is engorgement of the pulmonary circulation with dilatation of the veins and capillaries, resulting in passive hyperæmia and pulmonary œdema. These are followed by dilatation of the pulmonary artery and its branches, with atheroma of their walls.

Affections of the orifices and valves of the right side of the heart lead, by interference with the venæ cavæ and their tributaries, to venous hyperæmia followed by œdema. This is shown in the superficial parts of the body by anasarca, and in the internal viscera by cyanotic changes in the solid organs and catarrh of the mucous membranes. A tendency to coagulation in consequence of the reduced rapidity of the circulation leads to the formation of clots both in the cardiac chambers and in the venous channels. Embolic processes are found not only as the result of such coagula, but from vegetations on valves being carried on the blood stream. Hæmorrhage also may result as the result of engorgement in any of the blood vessels. It is, however, much more commonly found in the lungs, as a consequence of mitral disease, than in any other situation.

Symptoms.—With the exception of the phenomena revealed by physical examination, valvular lesions do not by themselves directly produce pathognomonic appearances. It is only when their secondary effects arise that there are any characteristic clinical features. The results of physical examination will be detailed under each particular valvular affection, but it may be advisable to mention some of the phenomena produced secondarily by valvular disease. In briefly reviewing such symptoms, it is impossible to do so otherwise than in certain groups. In doing this it must nevertheless be borne in view that many symptoms are at once subjective and objective, while it is also to be remembered that some appearances primarily belonging to one system of the body may be so connected with another group as to be separated from it only with difficulty.

Digestive functions.—These are often disturbed on account of the passive congestion and chronic catarrh arising from venous stasis. The common symptoms of such disturbance are nausea, vomiting, and diarrhœa, as well as, but less frequently, hæmatemesis and melæna. Jaundice is common as the result of backward pressure on the hepatic venous radicles. When jaundice is present, it is almost invariably associated with enlargement of the liver, in which there may be distinct pulsation. Amongst symptoms connected with the abdominal viscera, ascites is of common occurrence.

Blood.—The blood is extremely variable, but may be said to vary between two extremes, that of cyanosis, and that of anæmia. In the former condition the blood is dark coloured and of high specific gravity. The amount of hæmoglobin is remarkably increased, while the number both of the coloured and colourless corpuscles is largely augmented. In order to give concrete instances, it may be said that the specific gravity sometimes reaches 1070, the hæmoglobin 160 per cent., the red blood corpuscles 9,000,000 per cm., and the white blood corpuscles 16,000 per cm. My own explanation of this condition refers it to a compensatory process. From insufficient oxygenation of the corpuscles, they do not so thoroughly perform their functions as oxygen carriers, and do not yield so much oxygen to the tissues. It may be further mentioned that in cyanosis there is less metabolism and therefore less waste. The functions of the corpuscles being thus lessened, the tear and wear to which they are subjected are diminished, and the duration of their existence is prolonged, until a balance is struck between production and destruction. When anæmia is attendant upon valvular disease, there is diminution of the specific gravity of the blood, reduction of the amount of hæmoglobin, and lessened number of the red blood corpuscles, in most instances along with an increase in the number of the white blood corpuscles. In certain cases when there is at once anæmia and cyanosis, intermediate combinations may be observed. In regard to the glands, it may be said that the spleen is the only organ belonging to this category in which obvious alterations occur. When there is much backward pressure with enlargement of the liver, the spleen undergoes some increase in size, but this is not as a rule noteworthy. The thyroid gland may be enlarged or diminished in association with conditions accidentally linked with circulatory disorders, but since the association is purely adventitious, the subject need not be discussed.

Respiratory functions.—These show disturbances in many valvular lesions. The commonest symptom is dyspnœa. Breathlessness may only be found on exertion. It may occur in paroxysms, or it may be present without intermission. In almost all cases presenting dyspnœa there is at the same time cyanosis, and the essential cause of both conditions is deficiency of oxygen. Periodic respiration, or Cheyne-Stokes breathing, is a form of respiratory rhythm characterised by arrests of breathing. The stoppages occur periodically, and it may be said that there is a secondary rhythm imposed upon the primary rhythm of breathing; there is further, during the phase of activity, a gradual increase in the amplitude and acceleration of the rate of the respiratory movements up to a climax, after which there is a gradual decrease until complete cessation occurs. Along with periodic changes in the respiration, there are sometimes synchronous modifications in the volume and pressure of the pulse, in the size of the pupil, and in the state of the consciousness. Among other respiratory symptoms, yawning and hiccough are occasionally found in severe cases of cardiac failure from valvular disease. Cough may occur from bronchial catarrh, pulmonary congestion, pulmonary œdema, or pleural transudation. Hæmoptysis is one of the most characteristic pulmonary symptoms of cardiac disease, more particularly amongst those suffering from mitral affections.

The **integumentary system** may show the opposite conditions of pallor and cyanosis, while jaundice occurs when the liver is implicated. The amount of perspiration is subject to great variations, but with loss of compensation there is frequently an abundant cold sweat, probably from a paralytic condition of the vaso-constrictor mechanism. Œdema of the skin and subcutaneous textures is a common symptom.

The **urinary symptoms** of cardiac disease are tolerably uniform. The amount of secretion is diminished, its specific gravity raised, and its colour deepened. Copious deposits of urates are common. Albuminuria is frequent, probably from escape both by means of the Malpighian tufts and the intertubular capillaries. Blood often appears as the result of rupture of the capillaries, tube casts are frequently found and are usually of a granular or hyaline type, but not infrequently there are blood casts.

Generative functions.—Amenorrhœa is common in mitral lesions, especially along with anæmia; leucorrhœa and menorrhagia are often found from venous stasis.

The **nervous system** shows manifold disturbances in valvular lesions, particularly those of the aortic orifice. Sensory disturbances varying from slight uneasiness to overwhelming pain may be present. These may be accompanied by sensations of sinking or of fainting, or be accompanied by feelings of fluttering or trembling within the chest. When the sensory system is tested objectively, it is frequently found that there is hyperæsthesia over the areas to which the subjective sensations are referred. Headache and giddiness are sometimes found in valvular diseases, and the special senses may show considerable disturbance, usually in the direction of dulness, but occasionally exaggeration or even hallucination may be found. Sleeplessness and sleepiness are both met with, and there may be alterations in the mental functions culminating in unconsciousness or delirium.

Diagnosis.—It is unnecessary in this general introduction to Diseases of the Orifices and Valves to deal with questions of diagnosis, since the differentiation of the various lesions, whether single or combined, must be considered when treating of the individual diseases. Nothing, therefore, need be said in this place upon the subject.

Prognosis.—In estimating the probable future of chronic valvular disease, a number of factors require consideration. Amongst these the family history, sex, age, date of onset, occupation, habits, and surroundings, as well as the past medical history of the patient, furnish indications of importance. Over and above these different factors, it is necessary to remember that the nature of the lesion, its mode of origin, the length of its duration, and its effects upon the heart in particular, and the general system at large, are of vital importance. The prognosis as regards each particular valvular lesion will be considered in its appropriate place, and but little requires to be discussed in the present connection. It may be said, however, that from the length of time elapsing since the commencement of any valvular disease, it may be possible to predict the probable future of the lesions, since cases which have existed for some time without serious inconvenience will naturally be regarded with greater hopefulness. The influence of a valvular lesion upon the chambers of the heart is of significance. An undue tendency to dilatation at the expense of the compensatory process of hypertrophy is a harbinger of evil, while a tendency to cardiac failure is of grave import. Lastly, the effects of the lesion upon the general condition of the system must be estimated by the state of the digestion, of the blood, of the lungs, of the kidneys, and of the brain. When symptoms of disturbance in the functions of these organs are present, they render the prognosis somewhat more serious.

Treatment.—The existence of a valvular lesion does not in itself render any treatment necessary. It is only when valvular affections are associated with, or followed by, consecutive disturbances that it is necessary to interfere. Unfortunately, from the nature of the affections, prophylaxis, in the ordinary sense of the phrase, is scarcely applicable to valvular lesions. If preventive measures, in the strict acceptation of the term, are, as a rule, out of the question, there are nevertheless some matters connected with the management of valvular affections closely related to such methods. When there is any damage to the valvular mechanism, the entire circulatory system works at a disadvantage, and the very processes of compensation lead inevitably to disaster. It is therefore necessary to remove every baneful influence that might injuriously affect the circulation. The environment and occupation will require the most careful consideration with reference to their effects upon the individual, and such factors must be taken into account in relation to the age and sex of the patient.

Cases of valvular disease naturally fall into two groups—those with adequate compensation, and those in which it is insufficient. When compensation is sufficient, although watchful care must be exercised, it is well to avoid too much solicitude. Residence, in so far as possible, must be selected with a view to averting excessive vicissitudes of climate. The diet ought to be regulated, so as to maintain the strength while not taxing the digestive possibilities of the individual, and the amount of alcohol in particular must be definitely arranged. An occupation must be chosen, if possible, which will allow of sufficient open-air life, and the amount of exercise and rest is to be carefully determined. When compensation is not adequate, and the various symptoms of cardiac failure are present, they must be combated with the most sedulous attention. These symptoms depend one and all upon diminution of arterial pressure. One general scheme of treatment is accordingly applicable to almost all conditions of valvular disturbance, but this must of necessity be considerably modified in details to meet different cases. The regulation of light, air, exercise, rest, and sleep must be diligently attended to in every case, and the diet so arranged as to furnish adequate nourishment without digestive disturbances.

When there are evidences of serious cardiac failure, absolute rest is imperative in such a posture as will afford the greatest ease to the patient. It is often advisable to have two beds in the sick-room, one for use by day, the other by night. According to the conditions of the circulation, there must be regulation of the diet; in certain cases, where there is little or no œdema, but much digestive disturbance, a purely fluid diet will be indicated, while in others, with much œdema, the food should be concentrated. In most cases of cardiac failure one or other of the cardiac tonics will be found necessary. The only ones which need be mentioned are digitalis and strophanthus. Speaking generally, when rapid effects are desired, more especially in cases where the digestive system is irritable, strophanthus is to be preferred. If, on the other hand, œdema is excessive, and the renal secretion scanty, digitalis is of greater utility, especially in the form of the infusion. In such cases as have hepatic engorgement and digestive catarrh, small doses of blue pill, grey powder, or calomel will be found of the greatest importance. When œdema is alarming, the employment of concentrated saline aperients, along with such alkaline salts as the citrate or acetate of potash, which act upon the renal epithelium, is to be recommended. In my opinion, the employment of such means as the aspirator or Southey's tubes is to be postponed as long as possible. There can be no doubt that in the overwhelming proportion of cases when there has been recourse to such direct methods for removing fluid, their employment must be persisted in. In a small number, nevertheless, more particularly of cases in which acute heart failure has followed upon valvular disease, a return of compensation follows the employment of such measures. When profound cyanosis is present, the employment of oxygen is often of the greatest service, and when the symptom is obviously one indicative of extreme danger, general blood-letting should be resorted to without hesitation. The amount of renal secretion is of importance both in regard to quantity and constituents. Under the influence of the cardiac tonics, the diminished secretion as a rule becomes rapidly increased until a certain point has been reached far above the average, whence it diminishes to something like the normal amount. So long as this amount is maintained, the action of such drugs is entirely beneficial, and if the quantity falls below the average it may usually be taken as evidence of saturation. If albumin is present, it frequently disappears under the influence of the cardiac tonics, and the total amount of urea and its allies, diminished in consequence of the disease, approximates to normal. If the cardiac tonics are insufficient to increase the renal secretion, even along with salts of the alkalies, caffeine or theobromine may be usefully employed. When sleep is disturbed, as is often the case, simple remedies may suffice to get rid of the sleeplessness. Some warm mixture containing alcohol given at bedtime may be efficient, or one of the modern hypnotics, such as paraldehyde, sulphonal, or trional may produce the desired result. Not infrequently morphine is absolutely necessary, and there should be no hesitation about its employment, since it produces stimulant effects upon the circulation. When such serious cases have improved, and it may be said that this applies to other cases which are less serious throughout, there come to be questions with regard to exercise. This may be obtained in different ways: in patients who are too weak for any active exercise, massage and passive movements will be found of importance. In those who have still further improved, and in those whose condition is throughout more satisfactory, the use of Nauheim baths and resistance exercises may be enjoined; and when recovery has to a considerable degree occurred, active exercise carefully arranged should be taken advantage of.

AFFECTIONS OF THE AORTIC ORIFICE.

In the vast proportion of cases of aortic disease there is both obstruction and incompetence; since most lesions causing the former condition involve to some degree the cusps, and produce some retraction or deformity. Aortic obstruction without incompetence is more common than incompetence without obstruction, the explanation of which is that some lesions of the cusps and orifices do not interfere with the mechanism of the valves, while almost all lesions producing incompetence interfere with the exit of blood, and therefore produce obstruction. Incompetence is rarely caused by widening of the orifice itself, but some recent observations of Newton Pitt show that it is not unknown. Statistics upon the relative proportion of obstruction and incompetence are misleading, for the reason that in many cases where a double lesion has existed, the diagnosis recorded has been that of the affection which was most striking. Gillespie's statistics from the Edinburgh Royal Infirmary are as follows:—

Aortic obstruction	40
Incompetence	141
Obstruction and incompetence	230

These figures are far from according with my own experience.

Aortic Obstruction.

This lesion may be said to be absolute or relative; the orifice may be constricted so as to present less than its normal lumen, or, while the orifice is of ordinary size, a dilatation of the aorta beyond may produce physical signs closely resembling those which have their origin in contraction of the aortic orifice. In this connection, obstruction due to diminution of the lumen of the orifice will alone be considered.

Etiology.—Aortic obstruction is more frequently found in middle life and with advancing years, and it is much more common in men than in women. Occupations which produce long-continued severe muscular efforts are likely to cause it. Endocarditis, especially in its chronic form, accounts for a certain proportion of cases, but more important than endocarditis are sclerosis and degeneration.

Morbid anatomy.—The situation of the obstruction may be near the border of the cusps, or on any part of them exposed to the blood current. It may be at the line of their attachment to the aorta, or at the real origin of the arterial channel, especially close to the aortic cusp of the mitral valve. The nature of the lesion is very variable. It may be produced by vegetations, varying in size from insignificant granulations, like a millet-seed, to cauliflower-like masses larger than a full-sized pea. Such vegetations may undergo degeneration and calcification. On the other hand, the valves may be thickened along their margins with some retraction, often attended by degeneration and calcification. In such cases, from fusion of the cusps, the orifice may resemble a funnel with its apex projecting into the aorta. When obstruction is due to injury, either direct or indirect, one of the cusps may be found to hang freely from a small attachment. In addition to such lesions, it is not uncommon to find that the cusps are fenestrated. One and all of the lesions referred to may be attended by sclerosis of the aorta. In many instances there is some occlusion of the mouths of the coronary arteries from the arterial changes.

In simple aortic obstruction the wall of the left ventricle is found to be somewhat hypertrophied, with but little dilatation of the chamber. This, however, depends upon the duration of the case, and if of long standing, there is apt to be a greater degree of dilatation. In such cases the mitral orifice may become enlarged and lead to mitral incompetence, with dilatation and hypertrophy of the left auricle, followed by changes on the right side of the heart. When the coronary arteries are obstructed, there is apt to be some chronic myocarditis.

Symptoms.—There may be an entire absence of all subjective symptoms in aortic obstruction, but various degrees of cardiac pain are not infrequent. It is in aortic disease, more especially, that angina pectoris is found, and when present it may be accompanied by sensations of fluttering, fainting, and giddiness, along with the sense of impending death, and hyperæsthesia over the painful areas. Breathlessness is rarely complained of.

There is little to be seen on general inspection, except a slight degree of pallor, until cardiac failure sets in, when cyanosis may be present. When aortic obstruction is associated with arterial sclerosis, the superficial arteries stand out prominently, and manifest tortuosity with characteristic sinuous movement during pulsation. Palpation of the radial artery gives different results, according to the conditions of the wall of the vessel. If there be little or no sclerosis, so that the walls are yielding and elastic, a characteristic form of pulse may be determined; but if there is a considerable degree of arterial sclerosis, the vessel is more rigid and less elastic, so that any special features of the pulsation are lost. The pulse, in such cases as allow of its being thoroughly investigated, is infrequent, regular, tardy, and sustained, but these characters become modified on the appearance of cardiac failure. A sphygmographic tracing shows a tardy and moderate up-stroke or rounded summit, with obliteration to a considerable extent of the tidal wave and aortic notch. Sometimes a thrill may be felt in the larger arteries accompanying the pulsation. The præcordia in most instances presents no striking feature, but it sometimes happens that excessive pulsation may be seen in the left intercostal spaces within the cardiac limits. The apex-beat is almost invariably displaced to some extent downwards and to the left. Its impulse on palpation is, as a general rule, displaced downwards and outwards. It gives the sensation of a forcible and sustained pulsation, and may sometimes be accompanied by a systolic thrill. A thrill, however, is more commonly felt at the base of the heart, but in other cases it may be felt, not only over the entire præcordia, but throughout the whole chest. On percussion, the size of the heart is almost invariably found to be more or less enlarged. The right margin undergoes little alteration, but the left extends at once further outwards and downwards than is the case in health. The characteristic feature revealed by auscultation is a systolic murmur, which varies considerably not only in its characters, but even in its localisation. Its point of maximum intensity, as a general rule, is above the aortic cartilage, and it may be most distinct about the middle or even the upper part of the manubrium. The murmur in its most characteristic mode of conduction is carried upwards to the summit of the sternum and outwards along the clavicles, but also downwards by the sternum and ribs to a varying extent. It is also, however, propagated along the great arteries by means of which it may be carried to the vertex of the skull, and for a considerable distance along the arms and even the legs. The second sound in the aortic area presents many variations; most commonly it is increased in intensity, and when there is dilatation of the aorta, it may be greatly exaggerated. The first sound in the mitral area is usually lower in tone and longer in duration. It may also be somewhat rough in quality. The sounds produced on the right side of the heart are not altered, unless there are consecutive or attendant changes in the right ventricle.

Diagnosis.—There is not much difficulty in the determination of aortic obstruction. Even in cases where the pulse renders no aid, and there is no thrill at the base of the heart, the characteristic murmur and its mode of propagation are of themselves sufficient to prevent error. It may, however, be mistaken for dilatation of the ascending aorta, pressure upon the aorta by tumour, obstruction of the pulmonary orifice, and persistent ductus arteriosus. In dilatation of the aorta there is often increased dulness to the right of the sternum at the level of the second and third costal cartilages, which, however, may be absent. On auscultation in simple dilatation of the aorta, the systolic murmur is soft and blowing, but it is conducted in the same way as the murmur of aortic obstruction. The second sound is greatly accentuated in dilatation of the aorta, and gives a drum-like quality, whence it has been termed by Potain, *le 'bruit de tabourka.* This accentuation of the aortic second sound may be simulated in arterial sclerosis along with renal cirrhosis. The pressure of intrathoracic tumours upon the aorta near its origin gives rise to physical signs, closely resembling those of aortic obstruction, and if there be no communicated pulsation, the systolic murmur produced by the pressure upon the aorta may lead to confusion; but there is no alteration in the character of the second sound, and on percussion an area of dulness will be found corresponding to the new formation. Pulmonary obstruction has to be kept in view, since the aortic systolic murmur may be localised to the left of the sternum. In pulmonary obstruction the systolic murmur is never propagated into the great arteries at the root of the neck, which alone is enough to prevent any confusion between the two affections. In patent ductus arteriosus there is usually a thrill, which has its maximum intensity in the second or third left intercostal space, about 2 in. to the left of midsternum. This is distinctly later than the apex-beat, and is longer in duration than the thrill of aortic obstruction. On auscultation there is a loud murmur distinctly following the apex-beat, and continued beyond the second sound. This murmur is seldom conducted up the vessels to the neck.

Prognosis.—In itself aortic obstruction is the least serious of the valvular diseases. It is free from the tendency to sudden death manifested by aortic regurgitation, and its duration is probably more prolonged than that of any other valvular affection. This statement, however, must be qualified by recalling the fact that the lesions of aortic obstruction are apt to be complicated by interference with the coronary arteries, a subject to which attention has recently been specially directed by Kelynack.

Treatment.—In most instances of uncomplicated cases of aortic obstruction, no special treatment is required, but the patient should be enjoined to avoid all excessive physical and mental stress, while sufficient physical exercise and mental employment must be secured. It is probable that the use of iodide of potassium retards the process of sclerosis, and the long-continued employment of moderate doses should be adopted.

When cardiac failure ensues, as it is apt to do at last, the treatment must be such as has been already laid down.

AORTIC INCOMPETENCE.

As an isolated lesion aortic incompetence is rare, but, from the statistics already given, it is seen, in association with obstruction, to be one of the most frequent valvular diseases. Aortic incompetence is almost invariably brought about by some affection of the cusps, for the orifice is supported by such a resistant ring as to be difficult of dilatation. Quincke nevertheless suggested that it may occur as a purely functional disturbance, from stretching of the orifice, and Newton Pitt has collected some cases which appear to bear out this view.

Etiology.—The factors which produce incompetence are almost identical with those giving rise to obstruction. Endocarditis, produced by any of its causes, may be the starting-point of lesions of the cusps, leading to their inadequacy. It is, however, much more commonly caused by sclerotic changes, partly the result of syphilitic lesions, but also by long-continued stress and chemical agents, such as alcohol. It is the most frequent valvular lesion produced by violence, and the cusp which is most liable to rupture is the left posterior.

Morbid anatomy.—The most frequent lesions leading to regurgitation are thickening and shrinking of the cusps, in association with atheroma and calcification. These changes are often attended by dilatation of the aorta beyond its orifice, and by obstruction of the coronary arteries. Vegetations are found in a considerable proportion of cases, along with various deformities of the cusps, and in certain instances there is a fusion of the cusps at their free edges, so as to produce a small aperture sometimes shaped like a button-hole. In almost all of these cases there is therefore obstruction along with incompetence, and incompetence as an isolated lesion must be the result simply of shrinkage of the cusps or of stretching of the orifice.

The effects of aortic incompetence are shown first by the left ventricle. From the escape of blood backwards from the aorta, during each diastole, there is an increased amount in the ventricle, so that it becomes surcharged. It has thus a larger amount of blood to accommodate, as well as an increased amount of work to perform ; the cavity, therefore, becomes dilated as well as hypertrophied. In aortic disease the limits of hypertrophy are apt to be narrowed by the existence of coronary disease, which leads to degenerative changes in the muscular wall. When this is the case, dilatation is in excess of hypertrophy, and cardiac failure is the consequence. So long as the mitral cusps are competent, the circulation in the lungs is not implicated, and the right side of the heart is not affected ; but if, with a failing ventricle and incompetent mitral cusps, there is backward pressure upon the left auricle, the pulmonary capillaries are distended, leading to implication of the right side of the heart and the general venous circulation.

Symptoms.—There are but few symptoms, so long as compensation maintains the balance of the circulation, except in those cases in which the valvular mechanism has been disturbed by traumatism. In the great proportion of cases of aortic incompetence, therefore, the clinical features are latent. When the coronary vessels are implicated, the heart is apt to suffer, and the nervous mechanism prone to disturbance. Pain of an anginous type, a sensation of throbbing within the chest, breathlessness on exertion, and attacks of giddiness, sometimes with headache, may be experienced.

The skin, as a rule, is paler than in health, usually from an unfilled condition of the peripheral vessels, but sometimes from sclerosis of the arterioles. On closely examining the behaviour of the arterial circulation, a capillary pulsation may be observed on any part of the skin which has been reddened by pressure or friction. This is produced by the great oscillations of blood pressure in the arterioles and capillaries. It is often beautifully seen in the halo of urticaria, and it may be observed close to the lunules at the base of the nails. The fundus of the eye may reveal increased pulsation of the arteries. Excessive pulsation of all the superficial arteries may be easily determined. The radial pulse shows the striking characters which are usually associated with the name of Corrigan, but which were previously studied by Hodgkin, and even noticed by Vieussens. The vessel, as a rule, is far from full, yet the pressure may be above normal. The pulsation may be frequent or rare, according to the different circumstances that are present, but the rhythm is perfectly regular, and the pulsations are quite equal. Each pulsation is abrupt and large, but of short duration, falling away from the finger the instant when it reaches it. It is often termed the water-hammer pulse, since the sensation which it produces is remarkably like that given by the throb caused by that toy. A sphygmographic tracing shows an abrupt and steep ascent of the curve with a descent almost as swift. There may be a considerable tidal wave, but the dicrotic notch and wave are slightly marked. Occasionally, a direct venous pulsation may be seen in the peripheral veins. This is a gentle undulation, which distinctly follows the radial pulse.

Inspection of the chest shows the apex-beat to occupy a position further down and out than the normal, while the fact of a vigorous systole may be commonly seen from the very distinct movements of the præcordia. On applying the hand to the chest, it is usually found that the cardiac systole is more powerful, and that the diastolic recoil is also increased. At the apex the pulsation does not only give the feeling of increased strength, but it occasionally has a somewhat rough quality, and this may be followed by a characteristic diastolic thrill. The vibrations may be confined to the base or to the apex, but are more commonly felt over a wide area of the præcordia. The area of cardiac dulness is always increased, and it is lengthened in a vertical direction as well as widened horizontally. On auscultation there is a characteristic diastolic murmur. The quality of the murmur varies within wide limits. It may be perfectly soft and blowing in character, or it may be harsh and rasping, and it not infrequently assumes a musical character. Its maximum intensity is subject also to some variation, but it is commonly found at a lower level than the conventional aortic area. Sometimes it is louder to the left than to the right of the sternum. This diastolic murmur is conducted mostly in the direction of the xiphisternum and the apex of the heart. Most commonly it is not heard further down than the junction of the sternum with the ensiform cartilage, and it often fails to reach the apex. The murmur, however, is sometimes heard, not only throughout the whole chest, but it may be observed by a bystander without applying his ear to the chest at all. A presystolic murmur at the apex was described nearly forty years ago by Flint as occurring in cases of aortic incompetence without mitral obstruction. It has been described by so many observers that no doubt can be entertained of his accuracy, and it is commonly explained by the theory, that the regurgitant blood stream is brought to bear directly upon the anterior cusp of the mitral valve. On auscultation of the larger arteries, it is not uncommon to hear two murmurs, one immediately succeeding the systolic, and the other the diastolic phase of the ventricles.

Diagnosis.—But little difficulty is experienced in recognising aortic incompetence. The violent pulsation of the arteries, the character of the radial pulse, the presence of capillary pulsation, the hypertrophy of the left ventricle, and the diastolic murmur, with its characteristic mode of conduction, should render the diagnosis absolutely clear. It is not to be forgotten that the murmur may have its maximum intensity to the left of the sternum, a fact which might lead to the diagnosis of pulmonary incompetence. The condition of the arterial and capillary pulsation, and the state of the left ventricle, should render such a mistake little likely of occurrence. It will be referred to under the head of pulmonary valve disease. Some aneurysms of the first part of the arch of the aorta are said to cause not only a systolic but also a diastolic murmur. If this be the case, the aortic cusps are incompetent either absolutely or relatively, and no question of differential diagnosis therefore arises.

Prognosis.—Aortic incompetence is the valvular lesion of all others in which sudden death is most likely to occur, yet in many instances life is not only prolonged for many years, but it is attended throughout long periods by good health. The prognosis is dependent upon the cause and nature of the lesion, the presence or absence of other valvular affections, the integrity or implication of the arterial walls, the healthy or morbid condition of the kidneys, and the normal or abnormal condition of the general nutrition. When the disease has taken its origin in endocarditis affecting the aortic cusps solely, the outlook is brighter than when the lesion is of a degenerative type, since the latter is more likely to interfere with the coronary circulation. Uncomplicated aortic incompetence is less grave than that attended by other valvular affections. Degenerated arteries make the forecast less hopeful, since there is much likelihood of coronary disease. Lesions of the kidneys throw a great strain upon the damaged heart, and render the prognosis more gloomy. When the general nutrition is satisfactory, there is greater hope of the heart surviving the damage it has sustained, than when it is indifferent. When the hypertrophy balances the disturbance, if there is a distinct second sound in the cervical arteries, and if the pulse pressure is good, the outlook is as favourable as possible in this disease.

Treatment.—No special treatment is required, except the avoidance of over-fatigue of every kind, careful attention to diet, and cautious employment of alcohol and tobacco. When dealing with cases apparently of degenerative causes, the use of the iodides is of the greatest importance. Iodide of potassium or of sodium may be given in 5 gr. doses for long periods. It need hardly be added that in those cases attended by anginous attacks the iodides are of the highest value. When failure of compensation ensues, the treatment must be conducted in accordance with the general principles already laid down. Certain fears have been expressed as to the employment of drugs like digitalis and strophanthus, in consequence of the supposed evil of prolonging the period of diastole, and permitting regurgitation to exert its baneful influence on the left ventricle for a longer period. Such fears are chimerical, and the experience of every day shows that one or other of these drugs is imperatively demanded when cardiac failure supervenes upon aortic incompetence.

Aortic Obstruction and Incompetence.

Combined lesions at the aortic orifice are much more common than either obstruction or incompetence separately. All statistics show this, and for the reasons previously stated it is probable that the lesions in combination are relatively much more common than even those statistics show. The Edinburgh Royal Infirmary statistics show that out of 411 cases of aortic disease, 230 were diagnosed as combined obstruction and incompetence.

Etiology and morbid anatomy. — The causes of combined lesions at this orifice are the same as those concerned in production of its separate diseases. The structural alterations are similar to those already described, but, being in combination, when there are valvular changes producing obstruction, there is always evidence of inadequacy.

Symptoms and diagnosis.—The clinical features which result from double lesions at the aortic orifice are characterised by the mingling of the appearances characteristic of either lesion. The combined effects of the double lesions are, however, as a rule more severe, and the resulting symptoms are therefore more pronounced. Sometimes the effects of obstruction, at other times the results of incompetence, are predominant. As a general rule, the amount of dilatation and hypertrophy is equivalent to that which would be produced by aortic incompetence. From the relative amount of obstruction and incompetence, the enlargement of the heart has, however, wide limits of variability. The differential diagnosis must be conducted in accordance with the principles already laid down.

Prognosis and treatment.—Although double lesions produce a more serious state of matters than that which is found in aortic obstruction, the prognosis cannot be said to be graver than that of simple aortic incompetence. As the incompetence is the most serious factor in combined lesions, the treatment of these is essentially that of aortic regurgitation.

AFFECTIONS OF THE MITRAL ORIFICE.

The relative proportion of cases of mitral disease necessarily varies in different statistics. Referring once more to the Edinburgh statistics, out of 1914 admissions with valvular lesions, 1120, or 58 per cent., were cases of mitral disease, of which 597 were in men and 523 in women. As the total number of valvular cases in men was 1241, while that in women was 673, it follows that mitral disease constituted 48·1 per cent. of the total in men and 77·7 per cent. in women. As regards the relative proportion, amongst purely mitral cases—obstruction, incompetence, and combined lesions—there were in men 133 cases of obstruction, 342 of incompetence, and 122 of double lesions; while amongst women there were 171 cases of obstruction, 243 of incompetence, and 109 of double lesions. It seems probable that in these statistics the most prominent lesion is often returned as the diagnosis in cases of combined disease.

Mitral obstruction as an isolated lesion is by no means common, while mitral incompetence alone is much more frequently met with, since the causes which lead to it are so diverse. In the largest proportion of cases of mitral disease, the two lesions are associated together.

MITRAL OBSTRUCTION.

This lesion was the earliest valvular affection recognised during life by means of the characteristic thrill at the apex. Although the pathognomonic murmur was undoubtedly heard by Bertin, yet its significance was only discovered by Fauvel, with whom began the real clinical history of mitral obstruction.

Etiology.—As regards sex, all observations are at one in showing that this disease is more common in the female than in the male sex; and the Edinburgh Royal Infirmary statistics bear this out, by the fact that 133 males and 171 females were admitted in the five years. The disease is essentially one of early life, and the same statistics show the largest number of admissions to occur between the ages of 20 and 30. Mitral obstruction is more frequently the result of endocarditis than is aortic obstruction, and this factor is most commonly produced by rheumatism. In a considerable proportion of cases, the endocarditis arises from causes which cannot be determined, and Potain has suggested that a certain proportion of cases may be due to tuberculous endocarditis taking its origin in an infection carried from the lungs. In addition to endocarditis, degenerative lesions are often found.

Morbid anatomy.—The lesions causing obstruction are found either at the orifice itself, or upon the cusps—the latter being more common. In the former position, there may be vegetations or sclerotic processes; in the latter, while similar lesions may take place, there is frequently a union of the cusps, so as to form something like a funnel projecting downwards into the ventricle, pierced by a mere slit resembling a button-hole. In such instances the chordæ tendineæ are almost invariably thickened and shortened. Whatever may be the nature of the obstruction, there are certain definite consequences which follow upon the development of the obstacle. The left auricle, as might be expected, undergoes more or less hypertrophy, and that the hypertrophy stands in a direct ratio to the amount of obstruction, has been proved by the statistics of Samways. In a certain proportion of cases there is dilatation as well as hypertrophy, but this is chiefly a result of failure. The condition of the left ventricle varies very considerably. In a certain proportion of cases, no doubt as the result of the diminished amount of blood which enters it, there is some reduction in the thickness of its walls, but in many instances the left ventricle is hypertrophied. This latter condition is no doubt often in part due to backward pressure interfering with the passage of blood from the arterioles into the venous system, but it is possibly also due to interference with the aspiration of blood from the auricle into the ventricle. The right ventricle almost invariably undergoes some hypertrophy, usually with dilatation, the results of which will be detailed under the head of Tricuspid Disease. In most instances of mitral obstruction there is chronic venous stasis in the lungs, giving rise to an increase in their size, with brown induration. With failing compensation, œdema of the lungs is almost certain to occur, and it is often attended by hydrothorax. In this valvular affection also pulmonary hæmorrhage is a very common occurrence, and the various phenomena of embolism—cerebral, renal, or otherwise—are not infrequent. The abdominal viscera are marked by hyperæmia when the right heart fails, as will be described in the section on Tricuspid Lesions.

Symptoms.—The disease may be absolutely latent, giving rise to no clinical features save such as are revealed by physical examination. In the majority of instances, however, there is some interference with the pulmonary circulation, showing itself by breathlessness, which may be only present during exertion, or may present a real cardiac asthma, or even a persistent dyspnœa. Closely linked with this, when there is interference with the blood current, is cough and expectoration. The character of the sputum is usually very fluid and frothy, frequently tinged with blood, and often containing a considerable amount of it. It is in this disease, more than in any other valvular affection, that pulmonary hæmorrhage is apt to take place with free hæmoptysis. When the right side of the heart fails, as a result of mitral obstruction, there are the characteristic features which will be described as attendant upon tricuspid incompetence. There are sometimes subjective sensations in mitral obstruction, but when pain is present it has not the dominating features of angina pectoris, or its characteristic tendency to radiate towards the shoulder and arm. Sleeplessness, giddiness, and headache are frequent in mitral disease.

The appearance may be striking. A dusky malar flush is often seen, with pallor of the rest of the face, and a deep tint of the lips. Such appearances are frequently absent, and there is a common variety of the disease attended by profound anæmia. The pulse may show no special features. In an early condition it may be poorly filled and of low pressure, with perfect regularity and equality. Later, in the course of the disease, the vessel becomes more empty and the pressure still lower, while the rhythm is extremely irregular, and the pulse waves very unequal. Pulsation of the veins of the neck is often seen as the result of tricuspid implication. The apex-beat is apt to be diffuse, especially when the right side of the heart is involved. It is usually displaced outwards to the left. The hand may elicit no information when applied to the chest, but more commonly it reveals a diffuseness of the beat, and increased pulsation about the ensiform cartilage. In many cases a thrill at the apex is to be felt. It may be presystolic or diastolic in rhythm, or it may occupy the entire period elapsing between the second and the succeeding first sound. It is caused by the factors which produce the attendant murmur, and they will be more fitly considered in connection with it. At the base of the heart, the hand sometimes detects a very distinct impulse accompanying the second sound. It is best felt in the second left intercostal space. The area of cardiac dulness is sometimes unaltered. As a rule, however, its transverse diameter is increased in both directions. Auscultation shows some characteristic alterations in the rhythm and character of the sounds, as well as the presence of murmurs. The murmurs present may be of presystolic or of diastolic rhythm, or a long murmur may occupy the entire interval between the second and the first sounds. It is produced in the first case by the contraction of the left auricle, and in the second by the aspiratory action of the left ventricle, but both agents are probably assisted by the onward flow of the blood returning from the lungs. When the murmur occupies the entire interval between the second sound and the first, all these causes are doubtless at work. It is but right to add that the causation of the presystolic murmur has for long been subject of controversy. Ormerod, Barclay, Turner, Dickinson, Tripier and Devic, and Brockbank have urged that it is produced by ventricular contraction, but the overwhelming majority of teachers and writers follow the views of Gairdner, Balfour, and Fagge. Cowan and Gibbes have recently suggested modifications of the orthodox view. Such murmurs are sometimes entirely absent. Mitral obstructive murmurs are notoriously variable in their appearance, disappearance, and reappearance. In cases of simple obstruction without regurgitation, but without any disturbance of the right heart, there may be nothing save slight roughening of the commencement of the first sound in the mitral area. Mitral obstruction is often accompanied by accentuation of the pulmonary second sound, or by doubling of the second sound at the base of the heart, in which the pulmonic element is the more distinct.

In very many cases of mitral obstruction there are evidences of implication of the bases of the lungs—slight impairment of the percussion sound, with crepitations; or of the pleural sacs—dulness on percussion, with lessened breath and voice sounds.

When the right side of the heart fails, there are signs of backward pressure upon the systemic venous system, as will be described in the section on Tricuspid Disease.

Diagnosis.—When the physical signs are fully present, there can never be any difficulty in determining the presence of mitral obstruction. The thrill, the murmur, the accentuated pulmonary second sound, or doubling of that sound, render evidences quite unmistakable. It is a different matter when no murmurs are present; but even in such a case, a slight roughness of the first sound in the mitral area, with pulmonary accentuation or reduplication, may furnish sufficient evidence to justify at least a suspicion of mitral obstruction; and if there be proof of venous stasis in the lungs, or any implication of the pleural sac, this will be strengthened. If a murmur of regurgitation in the mitral area should accompany such physical signs, the diagnosis of a double lesion would be unavoidable. It has already been shown that a presystolic murmur has been recorded in several instances of aortic incompetence without any mitral lesion. It is accordingly difficult to say how far, in a case of undoubted aortic disease, associated with a presystolic murmur at the apex, this latter physical sign should be regarded as proof of mitral obstruction, or as an instance of the murmur first noticed by Flint. The condition of the pulmonary second sound, the state of the right side of the heart, and the position of matters in regard to the base of the lungs, might be of use. Obstruction of the mitral orifice has recently been described by Ewart and Rolleston in an interesting case of a pedunculated thrombus attached to the wall of the left auricle. The clinical appearances were identical with those of mitral disease. In such a case there could be no possibility of reaching an absolutely correct diagnosis.

Prognosis.—In uncomplicated mitral obstruction the outlook is not grave. When obstruction exists alone it is not usually of high degree, and there is therefore but little interference with the circulation. When obstruction is present to a greater extent there is usually regurgitation as well. The prognosis in such cases will be considered afterwards. Even in simple uncomplicated mitral obstruction, the prognosis must always be given with caution, as any extra stress may produce a break-down of the circulation. It is further to be remembered that embolism may occur, or acute endocarditis supervene upon the old lesion. When compensation is disturbed, the outlook is necessarily serious. Compared with other valvular lesions, it may be said that mitral obstruction is graver than aortic obstruction, but less so than mitral incompetence, while it is much less serious than aortic regurgitation.

Treatment.—The great aim of treatment in mitral obstruction must be to lessen the tendency to disturbances of the lungs; and while adopting the general principles applicable to the management of all valvular diseases, the principal care, as regards mitral obstruction, must be to prevent or remove any pulmonary complications. The avoidance of sudden changes of temperature should be enjoined; and when there is any complication of the kind, the patient should be placed in an apartment thoroughly ventilated, but of equable temperature, while steam, either simple or medicated, may be administered as an inhalation. One of the cardiac tonics, along with an ammoniacal stimulant, will be found in such cases of great utility.

MITRAL INCOMPETENCE.

Regurgitation at the mitral orifice probably never occurs under physiological conditions, and therefore the arrangements as regards the mitral cusps are entirely different from those which will be shown to obtain at the tricuspid orifice.

Etiology.—Incompetence of the mitral valve may occur, in the first place, as the result of changes in the cusps or in their tendinous cords—changes which may be endocarditic or degenerative in origin; or may more rarely be caused by traumatic agencies. It may also be produced by changes in the orifice itself, as the result of all conditions which enfeeble the myocardium, and thereby lead to relaxation of the muscular ring surrounding the orifice. It may further be produced by dilatation of the cavity of the ventricle itself, which leads to disturbance of the action of the papillary muscles and their tendinous cords. Mitral incompetence, produced by valvular lesions, is quite incurable, but when it is the result of myocardial changes it may be recovered from.

Morbid anatomy.—When due to endocarditic changes, the incompetence of the valves is usually accompanied by some contraction of the orifice. The appearances of the cusps in such cases resemble those which have been already described under mitral obstruction. The incompetence is produced by shrinking of the cusps, whereby they are rendered incapable of closing the orifice. When degenerative changes cause incompetence, there is less shrinking, and the changes observed are rigidity with slight contraction, often associated with fatty and calcareous deposits. In cases produced by injury, there is rupture of a cusp or of one or more of its tendinous cords. Mitral incompetence, produced in whole or in part by changes in the cardiac muscle, shows a widening of the orifice, or an enlargement of the ventricular cavity. It need hardly be added that in many cases several of these appearances are found in association with each other.

The effects of the incompetence are produced mostly on the left auricle and the right side of the heart. When there is a free regurgitation, the left auricle is at once dilated and hypertrophied, and in such circumstances the condition of the right ventricle is one also of dilatation and hypertrophy. With lesser degrees of regurgitation, there is always less dilatation and usually less hypertrophy. The left ventricle itself almost invariably shows some enlargement of its cavity and thickening of its walls. This at first sight may not appear very easy of explanation, but it is probable that, on account of the regurgitation into the left auricle, a larger amount of blood is poured back by the auricle into the ventricle during auricular systole, and the ventricle must have an increased amount of blood at the commencement of its systole. The pulmonary veins are dilated in free mitral regurgitation, and they, as well as the pulmonary artery and its branches, are often atheromatous. The lungs are enlarged, hyperæmic, and œdematous, with frequent hæmorrhages. When the right heart fails, and the systemic veins are involved, the changes occur which will be described in dealing with Tricuspid Incompetence.

Symptoms.—The affection is frequently latent. So long as the right heart maintains its integrity, the balance of the circulation is sustained, and no obvious disturbances are manifested. When compensation fails, a characteristic train of clinical appearances is developed. There may be uneasiness and weight in the region of the heart, but the commonest feelings are those of fluttering and trembling. Breathlessness is common, often accompanied by cough with watery expectoration. Sensations of faintness and giddiness with heaviness or pain in the head are of common occurrence, while insomnia and other brain symptoms are more likely to occur in this than in any other valvular lesion. When failure involves the digestive system, nausea, vomiting, and other symptoms take place.

The aspect of the patient suffering from this lesion often shows appearances like those which have been described under mitral obstruction. The dusky malar flush, the dark hue of the lips, nostrils, and ears, the arborescent capillaries upon exposed surfaces, and the congested look of the eyes, are at least as well marked in mitral incompetence as obstruction. When jaundice is united with such appearances, there may be a greenish hue of the complexion. Subcutaneous œdema of the dependent parts is frequent, especially at night. When there is such evidence of implication of the right side of the heart, the liver is often enlarged. The spleen also may be larger than in health, while ascites and albuminuria are also present. In still graver cases there may be some hæmatemesis and melæna. The pulmonary symptoms already mentioned are revealed by the presence of muffling of the percussion sound, and crepitations at the base of the lungs, or, it may be, absolute dulness and loss of the breath and voice sound.

The pulse is frequently unaltered, and in simple uncomplicated cases it presents no change, except reduction of pressure and volume. In graver conditions, and more particularly when associated with obstruction, the pulse is empty, its pressure low, its rate hurried, and its rhythm disordered. No morbid phenomena may be seen on inspection of the neck and chest. Venous pulsation, however, is not uncommon, from disturbance of the right side of the heart. The apex-beat is usually displaced outwards to the left, and is frequently diffuse. Pulsation, in many instances, may be seen in the second, third, and fourth left intercostal spaces, and epigastric heaving is a frequent occurrence. Palpation most commonly reveals a rather diffuse pulsation at the apex, often of somewhat less intensity than in health, and frequently followed by an extremely sharp impulse accompanying the second sound, which can be best felt at the base. Uncomplicated mitral regurgitation is rarely, if ever, accompanied by any thrill. Percussion shows an increase of the cardiac dulness both to the right and left. This increase is due in greatest part to the alterations in the right side of the heart. The constancy of the results of auscultation in this lesion afford a striking contrast to their variability in obstruction. As the characteristic murmur is never absent, so long as the heart is able to contract with even a very feeble degree of energy, a murmur which replaces, accompanies, or follows the first sound with its maximum intensity in the mitral area, is the chief feature of the lesion. Its character varies considerably. At times it is low, soft, and blowing, and at other times it is high, rough, and rasping. While too much stress must not be laid upon the character of the murmur, as a guide to the exact nature of the mitral lesion, it may, notwithstanding, be said that the murmur is usually higher in pitch and harsher in quality when the incompetence is produced by organic changes in the cusps attended by obstruction, than is the case when regurgitation results from simple relaxation of the orifice. The point of maximum intensity of the murmur is, as has been said, at or near the apex, from which point it is propagated in every direction to a varying extent; it is conducted to a greater distance in the direction of the axilla and scapula than elsewhere. Occasionally another murmur is heard in this affection, also of systolic rhythm, and heard with its greatest intensity at or near the second left intercostal space. It has been regarded by Naunyn and Balfour as due to the regurgitant stream impinging upon the interior of the left auricular appendix, but it is almost certainly of tricuspid origin. The second sound in the pulmonary area is, as a general rule, accentuated or doubled, but sometimes, although rarely, there may be a diastolic murmur of escape through the pulmonary orifice, resulting from high pressure.

Diagnosis.—Mitral incompetence can never present any serious difficulties in diagnosis, but it is not always easy to distinguish between the regurgitation due to a valvular lesion and that produced by simple dilatation of the orifice. When there is any evidence of obstruction as well as incompetence, the determination of a lesion of the cusps will be obvious. In this way, even in the absence of presystolic and diastolic murmurs, a roughening of the first sound and an irregularity of the rhythm may have some significant import. The statement, often current, that doubling of the second pulmonary sound is pathognomonic of mitral obstruction in cases of mitral disease, cannot be accepted.

Prognosis.—A forecast of the future in any case of mitral incompetence is not always a simple matter. The evidence furnished by physical examination alone may be misleading, as it is by no means easy to gauge the amount of regurgitation. As a general principle, it may be said that the louder the murmur of escape the more hopeful is the prognosis. The absence of any symptoms of backward pressure, a moderate degree of enlargement, an energetic cardiac impulse, and a loud first sound, are elements which would justify a hopeful prognosis, and the outlook is more favourable with a loud than with a feeble murmur. The presence of arterial pressure which is not too low, and pulsation of regular rhythm and not too great frequency, are favourable points. In addition to such special features, the existence of good general health, and freedom from pulmonary symptoms, will be helpful in shaping a favourable prognosis, while the various subjects discussed in the introductory section on valvular diseases, such as the age of the patient, the date of onset, and similar factors, must receive attention.

Treatment.—If the physician should be enabled to observe the development of mitral incompetence, as may happen during a febrile affection, he will be in a position to exert some influence upon the future of the disease. Absolute rest, so as to minimise fluctuations of blood pressure, and general tonic remedies, to aid the nutrition of the cardiac muscle, may lessen the mitral changes and obviate their consequences. In the case of mitral incompetence thoroughly developed, the general principles already laid down must be followed, and, in particular, care must be exercised so as to prevent as far as possible interference with the pulmonary circulation.

Mitral Obstruction and Regurgitation.

This combination is always due to organic disease; it is much more frequent in its occurrence than simple obstruction, but not so common as regurgitation singly, since incompetence has so many causes.

Etiology and morbid anatomy.—The lesions may be of endocarditic or degenerative origin, but the former is very much more frequently met with than the latter. It is unnecessary to consider the various factors at work in the production of these lesions—they are similar to those already considered under the separate diseases. Three distinct types of lesion are found on examination. Vegetations attended by some shrinking and retraction of the cusps form a well-marked group; another is found in those cases with thickening of the cusps and shrinking of the tendinous cords. In consequence of these lesions, there is a certain amount of obstruction and incompetence, from the impossibility of adaptation of the cusps to each other. The last class includes cases in which the cusps become fused, so as to form a slit, which from infiltration or deposition around it leads to incompetence as well as obstruction. The secondary changes are graver than in either lesion singly. The left auricle is implicated to a greater extent. There is more interference with the pulmonary circulation. The right side of the heart is involved in a higher degree, and the disturbance of the systemic veins is more serious.

Symptoms and diagnosis.—The clinical features resulting from the combined lesions show more or less blending of those which belong to each, but there are more marked symptoms when both lesions are combined than with either singly. The pulse is likely to become frequent and irregular at an early period. Venous symptoms in the neck and pulsation in the epigastrium are common. The cardiac dulness is exaggerated, and the combined murmurs may be heard. It must be borne in mind, however, that the murmurs of obstruction may fail, and the systolic murmurs of incompetence may be preceded by an accentuated or rough first sound. The pulmonary second sound is, as a rule, increased in intensity, and it may be doubled.

Prognosis and treatment.—The prospect is more gloomy in the case of combined lesions than of either singly. The greater severity of the effects leads to more disturbance of the functions connected with nutrition, and the course of the disease is shorter than in simple obstruction or simple regurgitation. It is unnecessary to say much as regards treatment, since it resolves itself into that already mentioned for the individual lesions.

AFFECTIONS OF THE PULMONARY ORIFICE.

Chronic affections of the pulmonary orifice constitute the rarest of valvular lesions, except in the case of congenital heart disease. Occurring so rarely as they do, these lesions do not furnish sufficient numbers to render statistics of any value. Amongst congenital valvular diseases, affections of this orifice are relatively frequent, but it is quite otherwise in regard to acquired pulmonary diseases, *i.e.* those developed after birth, for pulmonary obstruction and pulmonary incompetence are very rare. Vimont has with great diligence collected thirty-eight cases, in which the main feature of the lesion was obstruction, and Barié has brought together fifty-eight instances, the chief feature of which was incompetence.

PULMONARY OBSTRUCTION.

The remarks which follow are intended to apply, in so far as possible, solely to acquired pulmonary obstruction.

Etiology.—The causation is in many instances quite obscure, but the most common factor is endocarditis, arising in the course of various acute diseases. Rheumatism and the eruptive fevers are the most frequent causes giving rise to the endocarditis.

Morbid anatomy.—The most common lesion is produced by some fusion of the cusps, so as to produce a funnel-shaped structure with its apex pointing towards the artery. Instead of this, the cusps may be simply thickened and indurated as well as retracted. There may also be a vegetative condition, like that already described elsewhere. The obstruction is sometimes produced, however, within the walls of the conus arteriosus, in which there are deposits of fibrous tissue, and sometimes destructive changes involving the interventricular septum.

Symptoms.—A high degree of dyspnœa and cyanosis are commonly regarded as the great clinical features of this affection. To this it is scarcely possible for me to agree, as my own observations, although few in number, support the opinion expressed by Paul, that in acquired pulmonary obstruction there is only moderate cyanosis. It entirely depends, however, on the degree of obstruction. On the other hand, there is commonly clubbing of the fingers and toes and arching of their nails. Little tendency to venous stasis is found, and only slight subcutaneous œdema or visceral disturbance. There is evidence in acquired pulmonary obstruction, as in the congenital form, of considerable liability to pulmonary phthisis. The pulse has no special peculiarity. There may be venous phenomena in the neck, but they are not always present. No præcordial changes may be observed on inspection, but sometimes the apex-beat is further to the left, and there is epigastric pulsation. A systolic thrill is often felt at the base, especially to the left of the sternum. The cardiac dulness may be increased transversely. A systolic murmur in the second left intercostal space is the most important piece of physical evidence. The murmur is usually harsh, but varies considerably in its character. It invariably presents a superficial quality, and may be conducted over a considerable area of the chest, but is not propagated into the arteries of the neck.

Diagnosis.—The recognition of pulmonary obstruction depends largely upon the fact of a systolic murmur at the base which is not conducted into the cervical arteries. If, with clubbing of the fingers and some dyspnœa and cyanosis, such a murmur is present, it will raise a suspicion of pulmonary obstruction. It is often difficult at first to differentiate aortic and pulmonary obstruction. The maximum intensity of the systolic murmur of aortic disease is of comparatively little importance, but its mode of conduction is of the highest moment. It has already been said that the murmur of aortic obstruction may be heard, with its maximum intensity, to the left of the sternum, but the propagation of the murmur is significant. Patent ductus arteriosus can always be differentiated by the rhythm of its murmur, which is not accurately systolic, but begins after the first and is continued beyond the second sound.

Prognosis.—The prospect in pulmonary obstruction is unfavourable. Most of the recorded instances have died in early life, usually from pulmonary tuberculosis or cardiac failure.

Treatment.—The general rules for the management of valvular lesions must be followed, and the only point which requires to be emphasised is the necessity for watching over the condition of the lungs. If there be any symptom of bronchial or pulmonary trouble, warm, moist, medicated air, along with cardiac stimulants, must be adopted.

Pulmonary Incompetence.

The pulmonary valve may be incompetent in two different ways. Regurgitation may be allowed by means of structural alterations in the cusps, or may be permitted by stretching of the arterial orifice.

Etiology.—Acquired pulmonary incompetence due to valvular lesions is most commonly produced by endocarditis arising from acute rheumatism and eruptive fevers. It may also be produced by degenerative processes, causing sclerosis and retraction. The other form of incompetence, which has been the subject of observations by Steell, Duckworth, Barr, and myself, is produced by stretching of the orifice in consequence of increased pressure within the artery, such as frequently occurs in mitral disease.

Morbid anatomy.—The cusps are commonly thickened and indurated, as well as shrunken and distorted. They may have various vegetative and ulcerative changes upon their surfaces. The orifice is sometimes dilated, but it is more common to find it diminished. There is dilatation with hypertrophy of the right ventricle, and usually also of the right auricle. There may be such secondary lesions as are found in interference with the venous return, as will be discussed in dealing with tricuspid disease. In the relative or functional form of pulmonary incompetence, no morbid changes may be found on post-mortem examination, or the orifice may be found dilated while the cusps are healthy.

Symptoms.—It is difficult to estimate the nature of the symptoms produced by pulmonary incompetence, since the lesion is so rare, except in association with some other disease. Dyspnœa, cyanosis, and clubbing of the fingers are probably always present, and in consequence of the implication of the right ventricle there are the different features of venous stasis. The pulse is empty and compressible, venous phenomena are present in the neck, the transverse extent of cardiac dulness is increased, a diastolic thrill has been described in the second left intercostal space; a diastolic murmur with its maximum intensity in the second left intercostal space, propagated towards the apex along the left edge of the sternum, is characteristic. The quality of the murmur may be blowing or rasping.

Diagnosis and prognosis.—There should be no difficulty in the recognition of this affection, if aortic incompetence can be excluded. The position of the maximum intensity of the murmurs may be deceptive, and their conduction may further be closely similar. If the diastolic murmur can be heard at the apex, it is probably aortic; and if it can be heard over any of the great arteries, it is undoubtedly aortic. Capillary pulsation and the water-hammer pulse are strong points in favour of aortic disease, while cyanosis, dyspnœa, and clubbing of the fingers afford weighty evidence in favour of pulmonary incompetence. Regurgitation at the pulmonary orifice must always lead to a grave prognosis. Pulmonary complications are extremely common, especially when the disease exists in youthful patients.

Treatment.—The management of pulmonary incompetence must be specially directed with a view to obviate pulmonary complications, and if any should make their appearance they must be treated sedulously. The treatment of the affection otherwise is that of valvular disease in general.

Pulmonary Obstruction and Regurgitation.

The results of the double lesion, both as regards morbid anatomy and clinical features, are simply such as would naturally be expected, but in most instances the one or the other element preponderates, and therefore overshadows the characters, which are more or less blended, of the double lesion.

AFFECTIONS OF THE TRICUSPID ORIFICE.

In respect of frequency, the lesions of the right auriculo-ventricular orifice afford a striking contrast; while obstruction is one of the rarest, if not indeed quite the rarest, of valvular lesions, regurgitation without doubt is that most frequently found. As regards this orifice, statistics are useless—in the first place, from the great infrequency of the obstructive lesion as well as the difficulty attendant upon its detection; and, in the second place, because minor degrees of incompetence produce but little disturbance of the circulation, and therefore require to be sought in order to be found. How little reliance is to be placed upon statistics, as regards the valvular affections of the right side, may be appreciated from the fact that in Gillespie's statistics for five years, out of 1914 cases of valvular disease only fifteen were returned as lesions of the right side of the heart—a smaller number than may be found of cases of tricuspid incompetence in a single day's work in the outside department of a great hospital.

Tricuspid Obstruction.

This lesion has been anatomically recognised since the days of Morgagni, but it was first diagnosed during life and verified after death by Gairdner. Tricuspid obstruction has been more or less fully recorded in 154 cases, collected partly by Leudet and partly by Herrick, 114 of which occurred in women and thirty-two in men, while the sex was not recorded in eight. In thirteen of these cases the obstruction of the orifice was the only valvular lesion present. In all the rest it was accompanied by other valvular diseases.

Etiology.—In a considerable proportion of recorded cases, no causative factors could be discovered. Out of the 154 cases previously mentioned, rheumatism was recorded in fifty-one; it was doubtfully present in four; while chorea seemed to be a factor in two. Rheumatism was excluded in twenty-eight, and in sixty-nine no etiological facts were mentioned. Some of the acute febrile diseases are doubtless responsible for tricuspid obstruction, while certain of the other factors previously mentioned are no doubt operative.

Morbid anatomy.—The most common alteration observed is some fusion of the cusps, so as to form a funnel-shaped structure surrounding an orifice, at times so small as scarcely to admit the point of the little finger. Vegetations are often also present, and all of them may be followed by the deposition of inorganic salts. The secondary results have not been clearly studied, since the disease has, with few exceptions, been accompanied by other valvular disturbances. Some hypertrophy of the right auricle would naturally be expected, and as a rule it has been present, attended by a considerable amount of dilatation. The systemic venous system being seriously involved in all cases of marked obstruction, there are the anatomical features produced by backward pressure upon the abdominal viscera.

Symptoms.—From its usual association with some other valvular lesion, the results of tricuspid obstruction are difficult to estimate. Many symptoms which have been recorded are doubtless due to secondary changes in the right side of the heart, arising from left-sided lesions. Sometimes there is no clinical evidence indicative of a tricuspid lesion, yet, on the other hand, severe dyspnœa, with profound cyanosis and coldness of the extremities, have been seen. There may, along with these symptoms, be œdema of the lower extremities, scanty and albuminous renal secretion, with enlargement of the liver and spleen, as well as ascites. The veins of the neck may pulsate freely, or may be turgid and motionless. The radial pulse may be of average fulness and pressure without change in its rhythm or rate, but it has often been found empty, compressible, irregular, and accelerated. A thrill is occasionally felt with its maximum intensity just outside the left edge of the sternum. The transverse cardiac dulness is increased. The most characteristic clinical feature is a presystolic or diastolic murmur, heard at the junction of the fifth or sixth ribs with the left edge of the sternum. Occasionally, as in the case of the analogous mitral murmur, it begins with the second sound, and fills up the entire interval to the first sound.

Diagnosis.—Since the general symptoms of tricuspid obstruction closely resemble those produced by secondary implication of the right side of the heart, in consequence of mitral disease, the recognition of the lesion must depend almost entirely on the evidence of physical examination, and given the presence of the characteristic thrill and murmur, with their maximum intensity in the tricuspid area, the diagnosis may be established with a tolerable degree of certainty. In doubtful cases, a hepatic pulsation, preceding the apex-beat, is, according to Mackenzie, of diagnostic importance.

Prognosis.—The forecast of the future must be the result of careful consideration as regards each individual case, and must be rather based upon its association with other valvular lesions and the results of the cardiac disturbances upon the general circulation. Duroziez has shown that the average duration of life is directly proportional to the size of the orifice, but this cannot be of much importance to the practical physician, since there are never any grounds upon which to base even a guess at the size of the orifice.

Treatment.—The treatment of tricuspid obstruction must be conducted on the general principles already laid down for valvular disease, with special reference to the return of blood from the systemic veins.

TRICUSPID INCOMPETENCE.

This is the most common valvular affection. In spite of the adverse criticism to which their views have been subjected, there can be no doubt that the opinions of Hunter, Adams, and King are correct—a physiological regurgitation at the tricuspid orifice is easily produced. A thorough grasp of this conception is essential for the understanding of what may be termed pathological regurgitation, since what may be considered as physiological passes insensibly into pathological as regards leakage at this orifice. Tricuspid incompetence is more frequently met with than any other valvular lesion, but statistics entirely fail to bring out anything like its true numbers. It is so commonly found during the examination of patients who suffer from diseases of the most varied kind, that it frequently passes unrecorded.

Etiology.—The incompetence of the valve may be produced by some lesion of the cusps, such as endocarditis or degeneration, in the production of which the factors already cited as regards the other cusps play a similar part. But the incompetence is much more commonly brought about by some affection of the muscular substance of the heart. The factors which produce such changes are very numerous. It is often the sole cardiac affection present, yet it can scarcely ever be regarded as primary, but is almost invariably secondary to some other affection. It may be brought about by lesions of the pulmonary orifice, which in themselves are so rare as to be but seldom a cause of the lesion. Many diseases of the lungs, which interfere with the course of the blood through them, lead directly to tricuspid inadequacy. Mitral lesions are common causes of disturbance, but aortic diseases have in themselves no influence as long as the mitral cusps are competent and the left ventricle adequate. Such causes as renal disease, which interfere with the circulation, lead at length to implication of the right side of the heart and tricuspid incompetence. In addition to such mechanical interferences, there are many other causes more general in their nature, which produce their effects by acting on the muscular wall. Toxic influences of almost every class, every form of malnutrition, such as occurs in malignant disease, deficient absorption, or inadequate food, and every form of anæmia, may lead to the tricuspid regurgitation.

Morbid anatomy.—When the valves are at fault, there may be vegetations, thickening, fusion, and distortion of the cusps. It is, however, much more common to find that the valves are healthy, while the orifice is considerably enlarged; but in some cases of dilatation of the right ventricle there is little perceptible enlargement of the orifice, and the regurgitation must be produced by lack of adaptation of the valvular segments. Associated with these lesions are some alterations in the right auricle. It is usually dilated, sometimes to a great extent, and its walls are commonly thickened, but in old-standing cases they may be attenuated from malnutrition. The great veins leading to the right auricle are invariably dilated, and the valves of the veins of the neck are frequently incompetent. More outstanding effects are found in general venous stasis with subcutaneous œdema, serous transudation, mucous catarrh, and hyperæmia, leading to cyanotic atrophy of the solid viscera. This in time tells back upon the heart itself, which, by becoming the seat of chronic venous stasis, with consecutive degenerative processes, completes the vicious circle.

Symptoms.—The clinical features depend entirely upon the amount of regurgitation, and in minor degrees of incompetence the condition may be so latent as to be only discovered on physical examination. When the regurgitation is considerable, the features of the case are unmistakable. The aspect is cyanotic, or, if the liver is much implicated, there may be a greenish complexion, from the blending of lividity and jaundice. Dyspnœa is almost always present from interference with the pleura or the lung, or it may be simply from lack of aeration, due to deficient power of the right ventricle. Œdema of the lower limbs and ascites are common features, while on physical examination the liver and spleen may be enlarged. The renal secretion is scanty, albuminuria is frequent, often attended by hæmaturia and tube casts; hæmatemesis and melæna not infrequently occur. Headache, giddiness, and apathy make their appearance, while the night is troubled by insomnia or dreams. Such nervous symptoms as illusions, hallucinations, or even delusions, may be present. The circulatory organs themselves give very definite results. The radial pulse does not furnish any evidence of value, but it is usually of little volume and low pressure. The veins of the neck may show pulsation or manifest a graver symptom in persistent turgescence. Epigastric pulsation is a common appearance. There are sometimes also impulses in the third and fourth right intercostal spaces near the sternum. The apex-beat is further from midsternum than in health, but on account of its diffuseness it is often difficult to localise. The area of cardiac dulness is enlarged in both directions, but especially towards the right. The characteristic murmur of tricuspid incompetence is systolic in rhythm, with its maximum intensity in the tricuspid area. It is sometimes the sole evidence of the lesion, but, on the other hand, in cases of undoubted tricuspid incompetence, there may be no murmur and simply a weak or almost inaudible first sound. The murmur is in character soft and blowing, and it is conducted in every direction, so that considerable care must be exercised to distinguish it from murmurs in other areas. The condition of the second sound in the pulmonary area is by no means uniform. When tricuspid incompetence is produced by lesions of the left side of the heart or of the lungs, the accentuation of the pulmonary second sound, which precedes the development of the tricuspid regurgitation, becomes lessened. So long, however, as the pressure in the pulmonary circuit remains relatively increased, while it is relatively lessened in the systemic circulation, there will be pulmonary accentuation or doubling.

Diagnosis.—When characteristic venous appearances are present in the neck, they furnish sufficient evidence to lead to the suspicion of tricuspid incompetence, and if there be also a systolic murmur in the characteristic position, the recognition of tricuspid incompetence is easy. With a murmur alone present there may be a little more difficulty, and it is necessary to exercise care in its localisation, as well as to determine the size of the right side of the heart. If free venous pulsation is present in the neck, it may be accepted in default of all other evidence as strongly indicative of tricuspid incompetence, especially when it is of the rhythm shown by Mackenzie to be produced by a true ventricular wave.

Prognosis.—When tricuspid incompetence is obviously of muscular origin, and brought about by some transient disturbance, the outlook is excellent. It is far otherwise, however, if tricuspid regurgitation is a sequel to disease of the left side of the heart, or to some chronic pulmonary affection like emphysema. In cases of this kind, the right side of the heart can only recover if the primary affection undergoes amelioration.

Treatment. — The general management of valvular lesions is applicable to tricuspid incompetence, but the factors leading to the condition must be, in so far as possible, removed. When the affection is secondary to diseases of the left side of the heart, such must receive first consideration, and the same is true as regards diseases of the lungs. If the incompetence is the result of general conditions, such as anæmia or dyspepsia, appropriate treatment should be directed to these; and if the cause is cardiac strain from muscular stress, the exciting factor must be removed. In all cases cardiac tonics will be found of advantage, in addition to the other remedies which may be employed.

TRICUSPID OBSTRUCTION AND REGURGITATION.

Although they are more common than either obstruction or regurgitation, it is needless to dwell upon the combined lesions of the right venous orifice. From the standpoint of morbid anatomy, such lesions resemble tricuspid obstruction. In their symptoms they may closely resemble either obstruction or incompetence. As the obstruction is, however, frequently latent, the diagnosis of incompetence can often alone be made, and the lesions are frequently unsuspected during life.

G. A. GIBSON.

DISEASES OF THE MYOCARDIUM.

MECHANICAL CONGESTION.

In cardiac failure the heart itself is necessarily one of the viscera included in the area of mechanical congestion. The walls become of a deep red colour, dense and tough. Microscopical sections of the myocardium reveal the capillaries filled with blood; the muscular fibres pigmented, granular and atrophic-looking; and the connective tissue increased. It must be admitted that many pathologists refuse to allow of an interstitial myocarditis from simple passive hyperæmia, but it is difficult to avoid the conclusion arrived at.

This morbid condition of the cardiac wall, which was first particularly described by William Jenner, is of much more than pathological interest. Being the effect of failure of the heart upon the nutrition of its own walls, it represents the completion of a vicious circle of grave significance. The enlarged organ, labouring in the face of difficulties, is being worked on blood which is delayed in its flow and laden with the products of metabolism, in consequence of imperfect drainage of the veins. Congestion of the myocardium thus aggravates the dilatation in which it originated, and comes to be one of the chief causes of final failure.

It is, however, a condition which can be temporarily relieved by treatment. Congestion of the myocardium, along with that of the liver, kidneys, and systemic veins, yields to the medicinal and non-medicinal means which restore compensation. It will be referred to in connection with Dilatation followed by Failure.

ACUTE MYOCARDITIS.

Etiology.—Acute myocarditis commonly accompanies one of the acute febrile processes, such as rheumatism (when it may be associated with endocarditis and pericarditis), diphtheria, and scarlatina. A distinct kind of acute myocarditis is septic, in connection with injuries, particularly of bones and joints, chronic abscesses, stricture, ulcerative endocarditis, and the like. Very often no cause can be discovered. Traumatic inflammation of the myocardium has also been met with. The disease is much more common in males; and the septic kind usually occurs in boys.

Morbid anatomy and pathology.—Acute myocarditis is usually found as part of a pathological process which involves also the endocardium and pericardium. The left ventricular wall, including the septum and the papillary muscles, is the part most frequently affected, in the form either of a thin layer of inflamed tissue immediately beneath the lining and covering membranes, or of localised foci as large as a nut, pea, or a pin's head, or possibly of microscopic dimensions. The muscular tissue is of a dirty reddish yellow or buff, mottled appearance, swollen, soft, and friable. The cardiac chambers are dilated, particularly the left ventricle.

The localised form may proceed to abscesses, which are found either as such—small cavities with ragged walls, containing a dirty, puriform material, or as acute cardiac aneurysms, from rupture into one of the chambers of the heart. These cardiac abscesses are usually associated with ulcerative endocarditis and pericarditis; and the origin of the cardiac inflammation as a whole can be traced to one or other of the septic diseases just mentioned under etiology and attendant embolism.

Short of suppuration, either the localised or the diffused form of myocarditis ends in fibroid patches in the cardiac walls and their consequences.

Microscopically, acute myocarditis presents abundant infiltration of the interstitial tissues with leucocytes, micro-organisms, and blood—particularly along the sheaths of the vessels, which sometimes contain thrombi or infective emboli. The muscular fibres of the parts lose their striation, undergo nuclear proliferation (suggesting an attempt at regeneration), and finally degenerate, rupture, atrophy, and disappear.

The pathology of acute myocarditis is simple. It is commonly one of the local manifestations of an acute infective process. The *materies morbi* reaches the substance of the heart either by direct local extension from the surfaces, or through the coronary vessels. The severe symptoms are partly phenomena of the primary disease, partly of grave lesion of the muscular fibres of the heart, which fail before the arterial pressure, particularly when this is high, as it is in scarlet fever. At the same time it would be incorrect to refer acute cardiac failure in febrile or infective diseases wholly and solely to acute myocarditis. The cardiac nerves are unquestionably involved in some instances, and the muscular tissue as a whole is in a condition also of cloudy swelling, which is sometimes described as parenchymatous myocarditis. Multiple foci of infective myocarditis on a microscopic scale doubtless contribute to dilatation of the heart in some kinds of ulcerative endocarditis in chronic valvular disease. The pathology of acute cardiac aneurysm does not require to be discussed here.

Symptoms.—During the course of one of the diseases just mentioned, with its characteristic symptoms, the patient becomes restless, pallid, and peculiarly anxious in countenance. He complains of præcordial distress or actual pain, and of fluttering of the heart; the breathing is difficult; the pulse is very rapid (but sometimes infrequent), feeble, and possibly irregular; and vomiting may occur. After a short time the mental distress passes into delirium. The whole aspect of such a case is unmistakably "cardiac"; in other instances the prominence of paroxysmal dyspnœa, restlessness, lividity and cold sweats, suggests affection of the lungs. In a third set of cases cerebral symptoms are predominant—delirium, convulsions, headache, and finally coma. When these different clinical types of acute myocarditis have been accounted for, there remain a number of recorded instances of the disease in which no phenomena were observed, that pointed to involvement of the substance of the heart, apart from the endocardium and pericardium, or in which the symptoms of inflammation of the heart were obscured by those of the primary disease, of which they were usually regarded as complications.

The physical signs consist in a diffused feeble character (after brief increase) of the cardiac impulse, moderate enlargement of præcordial dulness, and alterations in the sounds, which become weak or small, the first sound finally being lost. The signs of pericarditis are commonly associated with these, and may entirely cover them for a time. The development of a blowing murmur is significant of endocarditis; in very rare cases it is produced by rupture of a parietal abscess inwards and the production of acute cardiac aneurysm.

Acute myocarditis runs a rapid course, and usually ends in death. Recovery no doubt occurs in rheumatic inflammation, limited to the layers of muscular tissue immediately beneath the covering and lining membranes of the heart, and sometimes even in the scarlatinal kind; but a favourable issue is quite exceptional in pyæmic myocarditis. The fatal termination may be sudden.

Diagnosis.—Acute myocarditis appears to be seldom diagnosed. It is lost, as it were, or overlooked, in the presence of the primary disease and of endocarditis and pericarditis. Acute failure of the heart in connection with these should arouse suspicion that the wall of the heart is involved, particularly marked disturbances of the rhythm and infrequency of the cardiac action, as observed in diphtheria. Pyæmic abscess of the heart is readily confounded with acute rheumatism, unless the history of injury of a bone, however slight (particularly in a boy), be duly regarded.

Prognosis.—The prognosis of acute myocarditis is always grave, and the prospect may be fairly estimated by the urgency of the cardiac symptoms and the state of the pulse and the circulation as a whole. If the disease be sufficiently developed to be diagnosed, recovery is almost hopeless. The liability to sudden death must be remembered.

Treatment.—The therapeutical indications in acute myocarditis, as far as they are derived from a consideration of its cause, are fulfilled by the treatment of the primary disease. Rheumatic carditis demands that salicylates be given with special care, their depressing effect being neutralised with ammonia, digitalis, strychnine, and alcohol in small repeated doses. Diphtheria, pyæmia, and septicæmia will receive proper attention.

A consideration of the pathological characters of the inflamed myocardium indicates the paramount importance of rest, as far as it can be secured for an organ of such activity. The recumbent posture must be maintained, and every kind of sudden movement avoided. Skilled nursing is of the last importance, particularly in diphtheria, where feeding, the management of the throat and nose, and subcutaneous injections disturb and exhaust the child, unless managed with gentleness and tact. Rest should be continued far into convalescence.

Whilst the heart is spared every unnecessary effort, its action, as estimated by the pulse and general symptoms, has to be steadily supported. The fulfilment of this indication requires judgment in the use of cardiac stimulants. Digitalis and its allies, ammonia and strychnine, are the chief means to be employed; but they must be given in small doses, well diluted, and at short intervals, lest sudden increase of the energy of contraction exhaust or rupture the inflamed fibres. The same consideration will control the selection and administration of food and stimulants; and flatulence must be averted, or removed by carminatives and gentle mercurial aperients. The same disposition to failure of the heart indicates the desirability of avoiding depressant drugs such as ipecacuanha, pilocarpine, etc., in the treatment of the primary disease. Præcordial distress is best relieved by the measures directed to the heart itself, but light warm poultices of linseed meal and mustard may also be employed. When failure is urgent, we must administer strychnine, ether, or alcohol subcutaneously, and employ other familiar measures for restoring the circulation.

CHRONIC MYOCARDITIS.

Syn., Fibrous Myocarditis; Fibrous Transformation; Fibroid Degeneration.

Chronic interstitial myocarditis occurs in a variety of forms. It constitutes the streaks and small areas of myocardial induration so often found on section of the heart, particularly in the papillary muscles, or underneath opaque patches of the endocardium and pericardium. Fibroid disease of the heart usually occurs near the apex of the left ventricle, and involves a considerable area and the entire thickness of the wall, which is variously puckered, pouched, or actually aneurysmal. Diffuse interstitial fibrosis of the myocardium, associated with a history of excessive indulgence in food and drink, has been described under the name of "Connective Tissue Hypertrophy of the Heart."

Etiology and morbid anatomy.—This disease is not always strictly inflammatory. Whilst it sometimes originates in the perivascular infiltration associated with acute endo-, peri-, and myo-carditis, it is also one of the results of syphilis of the heart, whether gummatous or vascular. In other instances it can be traced to coronary disease—degenerative, thrombotic, or embolic—and the subsequent atrophy or infarction. Rarely it arises from local injury. Increase of the connective tissue of the heart is sometimes referable to mechanical congestion of the myocardium.

Symptoms.—The symptoms and physical signs of chronic myocarditis are practically indistinguishable from those of chronic valvular disease with failing compensation, or of cardiac enlargement and degeneration in association with arterial sclerosis, Bright's disease, and fatty change. Irregularity, feebleness, and infrequency of pulse, angina pectoris, and dyspnœa have been prominent in some instances. Sudden death, without previous illness, is a characteristic termination when a considerable area of the heart is involved.

Treatment.—The treatment of this disease (if it were diagnosed) would depend on its pathological relations. Benefit is to be expected from a combination of iodide of potassium and tonic doses of digitalis and strychnine. In every instance the diet and the mode of life generally would have to be carefully ordered.

FATTY INFILTRATION.

Syn., Fat Heart; Adipose Heart; Cor Adiposum.

Excessive increase of adipose tissue upon and in the substance of the myocardium.

Etiology.—This disease is usually part of general obesity, and possesses the same pathological relations. It is more common in men, and after the age of 40. More rarely it appears to be a local change consecutive to muscular atrophy, as in essential atrophy of the voluntary muscles.

Morbid anatomy and pathology.—Cor adiposum is characterised by abnormal increase of adipose tissue in the usual situations on the heart, and its spread into the substance of the myocardium. In extreme cases the muscular tissue appears extensively replaced in bulk by fat. The organ is increased in weight, and its cavities may prove to be dilated. *Microscopically* fat cells are seen in greater or less abundance between the muscular bundles or individual fibres, which appear thin and widely separated. The muscle protoplasm is not necessarily changed, but may be atrophied or actually degenerated. In addition to the general obesity, the heart is obviously oppressed by the physical presence of fat, weakened by disturbance and atrophy of its proper fibres which permit dilatation, and subject to frequent functional derangement by the flatulence and disorders of blood pressure and metabolism associated with the adipose habit.

Symptoms.—Obese and corpulent persons habitually suffer some degree of dyspnœa and distress on exertion. When the heart is loaded with fat, there are added to these symptoms a sense of faintness, giddiness, feeble and irregular cardiac action, and præcordial oppression, especially under the influence of flatulence and gout. Physical examination of the chest and heart is very difficult in these subjects, particularly in women. As a rule the præcordial impulse is imperceptible, and the area of dulness indeterminate. The cardiac sounds are weak and small; occasionally a systolic murmur is heard in the mitral area. The pulse is weak and small. The condition lasts for an indefinite length of time, along with the many functional derangements usually associated with obesity. Sudden death is not unknown.

Diagnosis and prognosis.—Fatty infiltration of the heart is as a rule easily diagnosed by its associations. We must not, however, too readily conclude that the diagnosis "fat heart" perfectly satisfies the problem in every instance of cardiac distress that occurs in the obese. Fatty accumulation is but one of the morbid changes which affect the heart in these subjects. This is estimated not only by the cardiac symptoms and signs, but by the many other elements of the disease as a whole, such as gout, glycosuria, and Bright's disease.

Treatment.—The treatment of fatty infiltration of the heart involves the large question of the treatment of obesity. Here it is necessary to enforce the necessity of reckoning with the condition of the heart when we plan the more severe methods of reducing fatness. The diet must not include too many flatulent vegetables. Exercise is of great service, both for the disease as a whole and for the heart, but it must be intelligently planned and carried out, lest dilatation supervene. Both the Oertel and the Nauheim systems are successfully employed in *cor adiposum*. Special caution must be observed in recommending these patients to visit Marienbad or other baths, where the "cure" of obesity is practised.

A course of cardiac tonics, such as nux vomica and strophanthus, may be of service. Carminative draughts with alkalies, or stimulant mixtures containing ether and ammonia, will give relief in attacks of cardiac distress and failure connected with flatulent distension of the stomach.

FATTY DEGENERATION.

The term denotes fatty metamorphosis of the muscular fibres of the heart.

Etiology.—The disease is most common in males over 60, in whom the coronary arteries are degenerated in association with chronic strain, Bright's disease, or syphilis. An important group of cases in persons of less advanced age is traceable to changes of the blood itself, as in pernicious anæmia, phosphorus poisoning, chronic alcoholism, septic diseases, and cachectic states generally. Severe hæmorrhage may have the same effect. The change is more liable to occur when the heart is already enlarged, as in chronic valvular disease, arterial and renal degeneration, and after pregnancy. Fatty degeneration of limited areas may sometimes be traced to local interference with the nutrition of the walls of the heart by affections of the endocardium and pericardium.

Morbid anatomy and pathology.—The heart is usually enlarged, but its size and weight and the relative proportions of hypertrophy and dilatation of the walls vary with the cause of the disease. The appearances characteristic of fatty change are buff-coloured discolorations of the myocardium. These are best seen as uniform or mottled spots and patches on the inner surface of the left ventricle, particularly on the columnæ carneæ; but they may occupy any part of the heart, including the outer surface; and the change is often found microscopically when it cannot be otherwise differentiated. An area of degeneration can sometimes be accurately traced to obliterative disease of a branch of a coronary artery. In a small number of cases rupture occurs in the affected part. The substance of the heart as a whole is usually pale and friable, and in acute cases the change may be general or diffuse. Histologically the muscular fibres present fine particles of oil, arranged longitudinally between the elements from which they are derived, and which ultimately they entirely replace. Fatty degeneration of the heart originates either (1) in local or general interference with the quantity of blood supplied to, or taken up by, the active myocardium, or (2) in changes of its composition as the source of healthy nutrition and energy. The distressing symptoms, the characteristic progress, and the fatal issue are readily accounted for by enfeeblement of the cardiac walls.

Symptoms.—The characteristic symptoms of fatty degeneration of the heart occur chiefly in the form of intermittent attacks of acute faintness or actual syncope; præcordial oppression or pain, which may amount to angina pectoris; disturbances of the cardiac rhythm; a sense of weakness, unsteadiness, disposition to fall, and sometimes of impending death; sighing or Cheyne-Stokes respiration; nausea or vomiting, and coldness and dampness of the extremities. These phenomena may occur singly or in association, and may be of different degrees of severity and duration. An important feature of the disease is, that they recur on less and less provocation as it progresses, and therefore with increasing frequency. The patient first becomes aware of distress in connection with his heart and breathing on climbing stairs, hurrying to keep an appointment, or being exposed to excitement, whereupon a sense of faintness, fulness in the chest and throat, and irregular palpitation overtakes him: if he be walking he must stop and sigh deeply; and he may break into a cold sweat. After some time—it may be weeks or months—smaller and smaller acclivities, less and less rapidity of movement or exertion, induce similar or more severe symptoms, including pain. Finally, attacks come on even in bed, and in certain postures, particularly in association with flatulent dyspepsia. The patient may die in a seizure, which is then described as "syncope lethalis," or simply as "sudden death from failure of the heart." In these subjects death has occasionally occurred under an anæsthetic.

A variety of the attacks which demands special description, because of its great diagnostic importance, is characterised by coma or pseudo-apoplexy. We sometimes forget that amongst the "causes" or conditions underlying coma we must include fatty degeneration of the heart, and be prepared to meet with it. A patient complains of faintness or giddiness, may have a fit, and lapses into coma; more often he is found comatose—possibly in the street. In this condition he may die, or he emerges from it and dies very shortly, or he may recover for a considerable time, and pass through a series of similar attacks. The true nature of these cases is to be recognised by the association of coma with an excessively feeble or actually imperceptible pulse at the wrist.

In the intervals between the attacks the subject of fatty degeneration of the heart may enjoy fair health, provided he exercises due care. He presents the physical signs of vascular degeneration, of enlargement and feebleness of the heart, and possibly of valvular disease. The characters of the pulse vary greatly in different instances, with the associated pathological conditions; and often in the same patient at different times. Irregularity, intermittency, acceleration, and infrequency, slowness, smallness, and extreme feebleness are all met with in this disease, especially in connection with the seizures. Temporary mental disturbances sometimes occur. Lastly, this disease may exist without any symptoms to attract serious attention to the heart, the degenerated myocardium being only discovered after death. This often happens in acute cases where attention is diverted to the primary condition, such as pernicious anæmia or phosphorus poisoning.

Diagnosis and prognosis.—Fatty degeneration, when it occurs as a chronic process, may be difficult or impossible to differentiate from enlargement of the heart associated with arterial degeneration and Bright's disease, of which, indeed, it is often an incident. Its existence would be suggested but not proved by the age and life history of the patient, the state of the radial artery, the evidences of senility in the appearance and body generally, the occurrence of the symptoms which have been described pursuing their characteristic course, and the absence of the familiar phenomena of valvular disease, and, as a rule, of mechanical congestion of the viscera. Acute fatty degeneration of the heart is not regarded as other than a part of a grave pathological condition affecting all the organs. The prognosis of fatty degeneration of the heart is highly unfavourable in senile cases. The risk of sudden death must not be overlooked, and ought to be practically considered in connection with anæsthesia. When the disease is acutely developed, in connection with anæmia or the puerperal state, recovery may reasonably be anticipated.

Treatment.—In the chronic form of fatty degeneration of the heart, the principal indications are (1) to restore the nutrition of the coronary arteries and of the degenerated wall, and (2) to prevent and relieve cardiac distress. An attempt may be made to fulfil the first of these ends by careful revision of the patient's manner of living, including strict attention to the hygiene of advanced life in all its details. Daily exercise in the fresh air, either as gentle walks on level ground or easy drives, or both, and regulated by the patient's sensations; small meals of light but nutritious solids, rigidly excluding flatulent materials, and a small allowance of stimulants at meal-times, well diluted, are essential elements of the treatment. Smoking must be forbidden. The best medicinal remedies are combinations of iodide of potassium, carbonate of ammonium, nux vomica, and small doses of digitalis or strophanthus. Arsenic may be tried for a few weeks; and the effect of small doses of iron should also be tested with due caution.

Close attention to this *régime* may be successful in preventing præcordial distress, and at the same time the patient must be cautioned against sudden efforts and excitement. For actual attacks the best remedies are rapid cardiac stimulants, including ether and ammonia; combinations of these with carminatives, such as cajuput, lavender, cardamom, spirit of chloroform, etc.; and ordinary spirits in water. Hypodermic injections of strychnine or ether will be demanded when life is threatened. Angina pectoris is relieved with the nitrites.

The acute forms of fatty degeneration are treated as part of a general disease — anæmia, pernicious anæmia, phosphorus poisoning, etc.—the principal indications being to restore and purify the blood whilst the heart is protected from injurious strain. The importance of bodily and cardiac rest is obvious, in different degrees according to circumstances.

CARDIAC STRAIN.

This means damage of the walls of the heart, whether healthy or diseased, during muscular exertion. Damage of the valves, originating in exertion, is not discussed in this place.

Etiology.—Strain of the myocardium is the result of a stress falling upon its muscular and fibrous structures. This occurs either — (*a*) during a sudden violent effort, as in races, and strain is thus causally related to certain occupations, early adult life, and the male sex; or (*b*) from repeated or continued exertion, as in soldiers, miners, and other labourers.

Previous valvular disease conduces to strain, and so do disease and nutritional disorders of the myocardium, such as acute and chronic myocarditis, granular and fatty degeneration, fatty infiltration, etc. Old persons and the subjects of fever, anæmia, and gout are therefore peculiarly liable to cardiac strain. Distension of the stomach by a recent meal is sometimes a determining cause.

Morbid anatomy and pathology.—In persons dying during severe exertion, all the chambers of the heart are found choked with blood. The venous system is also loaded, and the lungs are engorged. If the injury have been survived indefinitely, the heart is dilated and hypertrophied on both sides, and evidences of previous endocarditis may be found on the mitral valve. In chronic cardiac strain the heart proves to have become enlarged from combined hypertrophy and dilatation of all its chambers, the latter in excess. The myocardium is the seat of granular, fibroid, and fatty degeneration. The coronary arteries are tortuous, thickened, degenerated; and their mouths are frequently found sclerosed. The mitral valves are thickened; the arterial valves often are remarkably free from change. All the ordinary characters of sclerosis are found in the arteries. In many instances the viscera and the serous and subcutaneous spaces present the familiar appearances of mechanical congestion and dropsy. The two main factors in the production of cardiac strain are—increased peripheral resistance, and increased influx of blood into the cavities.

(1) During severe muscular effort the arterial pressure rises and increases the peripheral resistance; the systolic discharge is insufficiently effected, that is, incompleted; and the residues which accumulate in the chambers dilate or stretch the walls. At the same time (2) the flow of venous blood into the heart from the muscles, through which the blood is poured in a larger and more rapid stream during their active contraction, is so abundant that it cannot be passed along into the arteries without leaving undischarged arrears, which add to the pressure on the elastic walls of the heart. So far only embarrassment has occurred. Extensibility prevents serious shock and strain. The heart feels distended and may ache, just as the arm or leg aches during unusual exertion. This degree of dilatation is physiological, and serves as a provision against something worse. The parts presently accommodate themselves comfortably to their more voluminous contents, and the man feels, as he puts it, that he has "got his second wind."

But the accumulation of blood and consequent pressure within the chambers may increase beyond the limits of elasticity. This is likely to happen during sudden as well as violent exertion when the resistance to efflux and the volume of influx are excessive; when the walls of the heart are weak or diseased, so that they fail in the effort to empty the chambers; and when the valves and openings have been previously damaged by disease, obstructing the outflow or permitting reflux. Under any or all of these circumstances the extreme and possibly sudden rise of pressure within the heart might cause either rupture of its fibres, spasm of the muscles with faintness and pain (angina pectoris), or cardiac paralysis (syncope). In most cases of effort, however, particularly if the heart have been previously healthy, these serious results do not occur. There happens, instead, incompetence or forcing of the auriculo-ventricular valves by the accumulation of blood within the ventricles; mechanical congestion of the veins behind; and extension or lengthening of the fibro-muscular structures. The accommodation of the accumulated blood in these several ways prevents actual rupture or other cause of sudden death, but the wall of the heart is damaged; and there remains over-stretching with dilatation, accompanied by a sense of fulness in the præcordia, characteristic physical signs, and incapacity for further exertion for a variable length of time—perhaps permanently.

Since exertion is only a relative term, it is obvious that strain of the heart in the widest sense comprises all conditions and cases in which the driving power of the chamber is at a mechanical disadvantage to the intracardiac pressure, that is, to the volume of its contents multiplied by the resistance ahead. The dynamical state in every form of valvular disease is one of increased strain. So is that of the subject of senile cardiac debility when climbing stairs or at stool, or possibly even when walking quietly on the level. And in one sense it must be said that cardiac strain occurs in every instance of dilatation with failure of the heart.

Symptoms.—Acute cardiac strain.—Violent muscular exertion is accompanied by certain phenomena, and certain sensations, particularly in a person out of "condition" or training. The face becomes livid and anxious; the veins of the neck and temples are full and pulsating; the eyes are prominent, the pupils dilated; heavy perspiration breaks out over the body; there is urgent dyspnœa in frequency and depth, and violent palpitation, both præcordial and epigastric, of regular or irregular rhythm, and greatly increased frequency. Subjectively there is a sense of fulness and distress across the lower sternum, which may amount to pain, spreading across the chest and into the neck and arms; a consciousness of want of breath, with inability to "get to the bottom of the breath," and of palpitation; and possibly faintness, general "muscular limpness," and mental confusion. If the subject is compelled to halt, or when he completes the effort, he may sink or fall down. He leans forward and presses his hands against his heart, or perhaps throws his head and chest back; opens his mouth; remains in the condition of distress and anxiety just described for a period varying from several seconds to a very few minutes; and he then emerges steadily from it, all the phenomena declining and disappearing together. If the præcordia be physically examined during this brief period, the area of percussion dulness and impulse is found to be increased, as well as that of audible heart sounds; and fugitive murmurs may be heard.

So far these are phenomena of nothing more than the temporary embarrassment of circulation and respiration attending severe muscular effort. But the result is not always evanescent and harmless. The other extreme is actual death, which probably occurs only in the subjects of previous cardiac disease. Short of a fatal issue, there may occur every degree of damage or disablement of the heart, particularly in the subject of previous cardiac disease; and it is to these instances that the term strain of the heart is applied. After an effort and its attendant phenomena, as described, the subject of cardiac strain in different cases finds that he is unfit for severe exertion as an athlete, or for the moderate exertion of everyday life, or perhaps even for gentle exercise, respectively. Muscular effort induces the same distressing phenomena as before. The professional athlete has to give up his occupation, the amateur his cycling or rowing or cricket. The older subject, whose myocardium had been previously weakened by gout or vascular degeneration, must halt again and again in going upstairs, and may even "feel" every acclivity on the road in walking, by the dyspnœa, præcordial fulness, disturbed or accelerated cardiac action, and possibly faintness which supervene, particularly after a meal, or if the health be impaired, or the heart otherwise disturbed by alcohol, tobacco, or emotion.

The course of acute cardiac strain will obviously vary much with the severity of the original damage, the age, occupation, and health of the subject, and other circumstances. Athletes often continue unfit for exertion for years, that is indefinitely, the heart being "irritable" when any extraordinary demand is made upon it. On the other hand, victims of cardiac strain, being compelled to avoid physical exertion, whilst they live carefully otherwise, may remain altogether free from symptoms of weakness of the heart. The physical signs connected with the heart may be those of dilatation with efficient hypertrophy. In other cases nothing abnormal can be discovered by clinical examination.

Chronic cardiac strain.—The subjects of chronic cardiac strain, referable to laborious mechanical occupations, such as the miner or the soldier, present the usual clinical phenomena of high pressure in the vessels and heart, as well as a disposition to the occurrence of embarrassment of the circulation and respiration on exertion. The radial arteries are increased in circumference (size) and length, tortuous, visible, locomotive, and thick. The left and possibly the right ventricle present the signs of enlargement, and various murmurs may be associated therewith. For a time there may be no symptoms of disability or distress, thanks to compensation. But after a time, varying in different instances, exertion or even exercise begins to induce dyspnœa (subjective and objective), palpitation, præcordial discomfort and faintness, as already described. The physical phenomena are now more significant of dilatation with failing hypertrophy. The pulse is of great variety, according to the condition of the heart vessels, lungs and kidneys. If the original cause continue, the condition drifts into one of confirmed cardio-vascular sclerosis and failing heart, with mechanical congestion and dropsy of the ordinary type.

Diagnosis. — There is rarely much difficulty in connecting the phenomena of acute strain of the heart with an unusual effort—whether absolutely excessive or in relation to the age and structural soundness of the patient. It may not be easy, however, to discover physical signs unequivocally corresponding to the cardiac injury. We have then to rely on the patient's statement and on the effect of a little muscular exertion in disturbing the frequency and rhythm of the heart, and producing other evidences of circulatory disturbance, during our examination.

The diagnosis of chronic strain of the heart is practically the diagnosis of arterial or cardio-vascular sclerosis, with or without valvular disease or Bright's disease, according to the phase which the affection may have reached.

Prognosis.—On the whole, a professional athlete, otherwise healthy, who has broken down during violent effort, and presents the symptoms and signs of cardiac strain, ought not to be encouraged in the hope of being able to resume the same occupation. Strain of a disordered heart, *e.g.* in the gouty, by moderate exertion, is less unfavourable prospectively, but severe exertion could not be safely undertaken in the future, although moderate exercise might be actually beneficial. Acute strain of a heart that is the seat of fatty degeneration might prove immediately fatal. The prognosis of chronic cardiac strain depends on many circumstances which are discussed under the heads of Diseases of the Arteries and Valvular Disease.

Treatment.—Strain of the heart is more easily prevented than repaired. The subjects of disease of the heart, or of the diseases which may be accompanied or followed by weakness of the myocardium, such as gout, should be cautioned against sudden or violent muscular efforts and muscular fatigue. At the same time it may be not only safe but positively desirable for them to employ moderate, carefully graduated exercise, to improve the nutrition of the heart, and thus diminish the disposition to strain.

The urgent distress attending the dilatation of the heart which constitutes acute strain is best relieved by rest and alcohol, the latter appearing to dilate the arteries whilst it increases the force and frequency of the heart, and thus promotes its rapid evacuation. The subsequent treatment of the condition when the heart is actually disabled, and of chronic strain, is very difficult. Rest for some weeks or months, cautiously followed by graduated exercise short of producing distress, is probably the best course to pursue. Small doses of strychnine and digitalis, and spare but nutritious diet, should be given at the same time; and all conditions unfavourable to cardiac nutrition and to cardiac action, as well as cardiac poisons such as tobacco and alcohol, must be studiously interdicted. With every possible care in these directions, the writer has more than once failed to benefit professional runners who had broken down in races; and he regards some cases of cardiac strain as practically incurable.

CARDIAC ANEURYSM.

Aneurysm of the heart consists in a cavity, sac, or localised depression in the walls of the heart, communicating with one or more of its chambers. In their origin and course cardiac aneurysms are either acute or chronic; and for the sake of clearness the two varieties will be separately discussed.

Acute Aneurysm.

Etiology and morbid anatomy.—This disease is usually developed in connection with acute endo- or myo-carditis. In the course of ulcerative endocarditis a limited portion of the myocardium—most frequently of the base of the left ventricle, including the septum—is invaded from the surface; and the tissues, infiltrated with septic organisms, exudation, and blood, yield before the intracardiac pressure. In other instances the initial lesion is in the depths of the wall, in the form of acute myocarditis, abscess, or infarction from coronary embolism or thrombosis (*myomalacia cordis*); and a sac is formed by the affected part passively yielding to pressure, or suddenly rupturing into the cardiac chamber.

Symptoms. — The existence of acute cardiac aneurysm can very rarely be ascertained clinically. The phenomena and course of the disease are those of the pathological condition in which it originates and of which it is an incident. Even the sudden development of a cardiac murmur and embolism of the viscera, suggestive of rupture of an inflammatory focus into the ventricle, are far more likely to be due to ulcerative endocarditis.

CHRONIC ANEURYSM.

Etiology and morbid anatomy.—This form usually originates in fibroid disease of a limited area of the cardiac wall, which gradually becomes sacculated under the influence of the blood pressure. It is thus one of the results of chronic myocarditis, elsewhere described. Chronic cardiac aneurysms are of all sizes—from a bean to a cocoa-nut. They may form tumours on the surface of the organ or lie concealed in the substance of the wall, betrayed post-mortem by the opening into the chamber. The contents are blood clots and fibrin. The left ventricle near the apex is by far the most common seat of the disease. Rupture may occur into the pericardium or into another cardiac chamber.

Symptoms.—The symptoms which attend aneurysm of the heart are the same as those of valvular disease—præcordial pain and distress, palpitation with disturbance of rhythm, faintness, or syncope. Sudden death is relatively common. The physical signs of most value for the establishment of a diagnosis (which is most difficult and seldom reached) are anomalous enlargement of the præcordial dulness, a peculiar whizzing sensation conveyed to the hand laid on the præcordia, and various murmurs. Occasionally there is tumour.

The treatment of the condition, if its presence were ascertained, would not be different from that of other forms of degenerative disease of the heart.

SPONTANEOUS RUPTURE.

A part of the wall of the heart which has been weakened by disease, particularly of a degenerative kind, may be torn across by sudden contraction of the healthy fibres. In three-fourths of the instances the lesion involves the left ventricle—usually its anterior wall. It may be complete, permitting extravasation of blood into the pericardial sac; or incomplete, constituting a rent or crack of the myocardium, whether externally, internally, or in its substance. In 77 per cent. of Quain's collected cases, fatty degeneration was said to be the cause of the diminished resistance of the cardiac tissue, but many cases originate in softening from infarction. The lesion, which is a very rare one, is almost confined to persons over 50 years of age.

The phenomena of rupture of the heart are very striking. During effort or violent excitement, a person who may or may not have been known to be the subject of cardiac disease suddenly complains of intense præcordial distress, becomes collapsed, and may die in a few moments. In other instances life has been prolonged for hours or even days, the symptoms continuing; in these instances rupture was probably gradual, or incomplete at first, or it involved the septum.

Rupture of the heart is almost invariably fatal, yet cases have been recorded in which the patient has recovered. In one of these, death was caused some years afterwards by another rupture. If the patient do not die immediately, his sufferings should be relieved by means of a hypodermic injection of morphine.

HYPERTROPHY.

This is an increase of the substance of the heart by enlargement and multiplication of its muscular fibres. Hypertrophy may involve one or more of the cardiac cavities or the whole heart. It is often associated with dilatation, but we will first treat of it here in its pure or simple form

Etiology.—Hypertrophy of the left ventricle is a result of aortic obstruction, of congenital coarctation and slow compression of the aorta, of chlorosis, atheroma and arterial sclerosis, and of Bright's disease. Hypertrophy of the right ventricle originates in pulmonary obstruction (very rare), chronic pulmonary affections, especially emphysema, and mitral and other diseases of the left side of the heart. Hypertrophy of the heart as a whole is produced by pericardial adhesion, by chronic myocardial diseases, by persistent muscular exertion or nervous excitement (Graves's disease, tachycardia), and by the continued action of cardiac poisons, such as alcohol, tobacco, tea, or uric acid. It also occurs temporarily in pregnancy. The relation of hypertrophy to its causes might be otherwise expressed by connecting it with laborious occupations, strain, the male sex, adult age, gout, certain habits, etc.

Morbid anatomy and pathology.—Simple hypertrophy of the heart is characterised anatomically by increased bulk, firmness, and weight of the organ, and by increased thickness of its walls on section. Naturally these changes are more appreciable in the ventricles; and various alterations in outline are observed (particularly in sections), according to the distribution of the hypertrophy in the different chambers. The columnæ carneæ are also thickened, particularly in the right ventricle; so are the musculi pectinati of the auricles, the walls of which are normally so thin that hypertrophy is difficult to estimate accurately. Simple hypertrophy of the heart, or of one of its chambers, is not disease but a process and a state of physiological repair, developed and maintained conformably to the laws of nutrition. Being an accurate automatic response to difficulties in the circulation, hypertrophy of the heart naturally removes morbid phenomena due to these; or if it be developed concomitantly with them—for example, at the end of acute endocarditis—it serves to prevent their evil results. Its effect is therefore compensation. Both its establishment and its maintenance depend on certain well-recognised conditions: an abundant supply of pure blood, wholesome nervous influences, and a reasonable demand for force; and failure of compensatory hypertrophy can always be traced to a break-down in one or other of these conditions, which may or may not be remediable.

Symptoms.—The clinical characters of hypertrophy of the heart are intimately associated with those of the morbid condition which accompanies and has provoked it. Hypertrophy is best studied as it affects the left ventricle in Bright's disease. When pure and perfect, it gives rise to no phenomena of distress or discomfort. On the contrary, the development of hypertrophy is found to remove or prevent instead of producing disturbances of the circulation, and to be the chief means of compensation for structural lesions. Palpitation, headache, epistaxis, and finally cerebral hæmorrhage, which occur occasionally in connection with hypertrophy of the left ventricle, are results of high arterial tension originating in renal disease, not in the hypertrophy itself, though no doubt they may be maintained by it. Such symptoms have sometimes been loosely described as those of "excessive hypertrophy"—which probably has no existence. It may also be safely asserted that the symptoms which occasionally accompany hypertrophy of the left ventricle in aortic obstruction, such as præcordial discomfort, faintness, and pallor, are produced by insufficiency, not excess, of muscular force, and that dyspnœa, cough, lividity, and the familiar signs of mitral disease are least marked when hypertrophy of the right ventricle is best preserved.

Physical signs.—The physical signs of hypertrophy of the left ventricle consist chiefly in visible and palpable præcordial bulging; a well-defined, localised, thrusting apex-beat, located downwards and outwards from its normal situation; moderate increase of transverse percussion dulness, and little or no appreciable increase of vertical dulness. The auscultatory signs vary with the lesion on which hypertrophy is developed. In a typical case of chronic Bright's disease, with pure hypertrophy of the left ventricle, the first sound is low and dull in the mitral area and weaker still at the bases, whilst the second sound is loud and ringing in connection with the high tension. The characters of the pulse depend upon the nature of the primary disease. Occasionally the arterial wall becomes thick and tortuous as a direct effect of the cardiac enlargement, which strains and lengthens it.

Hypertrophy of the right ventricle is characterised physically by bulging of the lower end of the sternum and its neighbourhood, and probably epigastric pulsation, displacement of the true apex-beat somewhat to the left and downwards, and increase of the transverse percussion dulness towards the right (including the lower end of the sternum), and slightly towards the left also. The auscultatory signs are various, according to the cause of the hypertrophy. Usually both first and second sounds are loud in the tricuspid area, and the second sound is accentuated at the pulmonary base, unless this region be covered with emphysematous lung. The pulse has no fixed characters; but it is usually small and poor in comparison with the vigour of the epigastric pulsation.

Course.—Hypertrophy quickly supervenes on a lesion which disturbs the circulation. In the course of a few weeks its physical signs may be appreciable after endocarditis. It continues indefinitely along with its cause, being permanent in ordinary valvular disease, temporary when evoked by passing strain or nervo-muscular excitement. Ultimately, in most cases, it declines in efficiency through failure of nutrition or increase of the cause, or both; and dilatation from failure supervenes, as we shall presently see. From this condition the hypertrophied heart often can be rescued. Such loss and recovery of compensation may be repeated indeed several times, until finally there is no response to treatment, and irremediable dilatation ensues.

Diagnosis.—Certain difficulties may attend the diagnosis of hypertrophy of the heart. The condition is obscured by concomitant affections of the chest, particularly emphysema, pleuritic effusions or hydrothorax, pericarditis, and deformities of the parietes. The discovery of the physical signs is often a difficult matter in women with large mammæ and in stout middle-aged or old men—a class of patients in whom the determination of hypertrophy is all-important. On the contrary, hypertrophy is simulated by displacement of the heart (particularly towards the left), from whatever cause, by violent action due to excitement or exertion, by the abnormal pulsations and dulness of aneurysms and new growths, by pericarditis and patches of basic pleurisy and fibrosis, and by bulging of the præcordia, "pigeon breast," and some other deformities. We must not forget that in children the apex-beat lies beyond, in, or just within the left vertical nipple-line, in the fourth or fifth interspace, according to age and growth of the diameters of the chest.

The diagnosis of hypertrophy from the other forms of cardiac enlargement will be presently described. In every instance the diagnosis of the existence of hypertrophy is to be made by intelligent attention to the physical signs and attendant circumstances. This being accomplished, the cause of the hypertrophy must be carefully ascertained.

Prognosis.—The prognosis of hypertrophy of the heart is estimated by attentive consideration of the pathological cause which has induced it, and of the conditions under which it is being carried on. Hypertrophy and the associated compensation are likely to be preserved as long as the demand upon the heart for force remains moderate, the supply of blood through the coronaries abundant and healthy, and the nervous impulses reaching the myocardium wholesome. The prospect that hypertrophy will remain good and sufficient not only varies, therefore, with the lesion and its cause, and with the social position, sex, and circumstances generally of different patients, but changes with the age and other changing conditions of the same patient at different times—in childhood, at puberty, during education, the incidence of the effects of bodily and mental strain, free-living, alcoholism, gout, senile degeneration, and so forth. Each period of life is indeed beset with its own peculiar dangers to the compensated heart.

Treatment.—Although hypertrophy of the heart is a purely conservative process, the need for it, and the demand on local or general nutrition which it involves, should be removed, if possible. This object can rarely be accomplished, valvular lesions being as a rule permanent and inaccessible; but persistent excitement and exertion, and cardiac poisons such as alcohol, can be dealt with; and the high tension of renal disease, whilst it cannot be permanently removed, can to some extent be controlled. The management of the renal, vascular, and cardiac conditions and relations in chronic Bright's disease with hypertrophy of the left ventricle, is one of the severest tests of the therapeutic art.

When the cause of hypertrophy is fixed and beyond our reach in the valves, pericardium, or vessels (and indeed in every instance, subject to the previous considerations), the indication is to promote it, that is, first to establish it and then to maintain it. The conditions of efficient hypertrophy have already been mentioned. The blood which reaches the myocardium must be abundant, rich, and free from excrementitious materials—a condition somewhat difficult to secure in chronic valvular disease, and still more so in renal disease. The coronary arteries must be preserved against degeneration (like the circulatory system as a whole) by attention to bodily hygiene. The nervous impulses which reach the heart from the centres, and reflexly from every part of the body, have to be controlled in all the affairs and functions of daily life, as far as this is possible—an end which neither advice nor care can by any means always attain. Much more can be done in hypertrophy by reducing the unnecessary mechanical work upon the heart, *i.e.* by preventing over-exertion and securing a sufficient amount of well-ordered rest.

In a word, the subject of hypertrophy of the heart must observe the rules of hygiene as faithfully as circumstances permit. Unless the primary disease present other indications (*e.g.* Bright's disease), he must enjoy an abundant, nutritious, and digestible diet; attend scrupulously to the action of the bowels, kidneys, and skin; clothe warmly; avoid exciting or worrying and laborious occupations; and have moderate but at the same time regular and sufficient exercise. Hypertrophy of itself calls for no medicinal treatment; but much can be done in this as well as other ways when hypertrophy proves insufficient, as will be discussed under the head of "Dilatation with Failure."

DILATATION WITH COMPENSATORY HYPERTROPHY.

Syn., Dilatation from Over-filling.

This is an increase in the capacity of one or more of the cardiac chambers associated with efficient hypertrophy of the walls.

Etiology.—The causes of this form of enlargement of the heart are valvular disease or injury, malformations, adherent pericardium, and strain. It is typically met with, affecting the left ventricle, in aortic incompetence and in mitral incompetence; and, affecting the auricles, in incompetence of the auriculo-ventricular valves.

Morbid anatomy and pathology.—The heart is enlarged to the eye, and heavier, weighing from a few ounces above the normal to as much as 40 and even 60 oz. The outline is more rounded than normal; the cavity or cavities are capacious and saccular; the columnæ carneæ appear flattened. Therewith the muscular walls are thickened from true hypertrophy, although the change may not be striking in relation to the dilatation. The condition of the muscular tissue varies with the origin and duration of the enlargement, and with the mode of death. In dilatation of the heart of this kind, we meet with a new factor of enlargement, namely, over-distension of the chamber. This occurs during diastole, by its receiving an excessive charge, which overstretches its elastic walls and forcibly increases its capacity. Such over-filling occurs in either of two ways. First, the chamber may be charged from two sources, as is the case with the left ventricle in aortic incompetence and with the left auricle in mitral incompetence. Second, the chamber may receive an excessive charge from the chamber or vessel immediately behind it, as is the case with the left ventricle in mitral incompetence, and just as another part of the circulatory system, the aorta and the whole arterial tree, is over-filled and over-stretched by receiving the contents of the dilated left ventricle in aortic incompetence. "Relative" over-filling of the heart, as it occurs during muscular effort, is described under "Dilatation with Failure," and "Strain."

It will be observed that this dilatation, as it occurs in connection with valvular disease, is a purely mechanical effect of valvular incompetence, and inevitably follows it. If the valvular lesion be permanent, as is usually the case, the mechanical dilatation is persistent or permanent too.

Meanwhile, however, the excessive charge does something more than over-distend and dilate the chamber. Involving as it does an increased volume and weight of blood to be discharged, it evokes more driving force in systole; and the persistence of this increased work leads to concomitant hypertrophy, accurately proportioned to the increased work or load, that is, to the dilatation. The result of this dual process of dilatation and efficient hypertrophy is compensation of the valvular defect, which is maintained indefinitely, provided the conditions of hypertrophy continue to be preserved. Theoretically there are no symptoms of disease. Still, the over-filling and under-filling respectively of the chambers, arteries, and pulmonary vessels, in the different forms of valvular disease, compel or render inevitable certain phenomena of disturbances of distribution of blood, such as pallor and possibly faintness on the one hand, lividity and dyspnœa on the other. The balance of the associated processes of over-filling and efficient emptying is also more easily disturbed than the simple balance between increased resistance and increased force met with in pure hypertrophy.

When disturbance does occur, as the result of exertion, nervous strain, or defective nutrition, compensation is interrupted, and failure ensues, as will be presently described under "Dilatation with Failure." But compensation may be regained, and this not once only but again and again, provided the conditions of hypertrophy are restored. The pathology of dilatation with efficient hypertrophy as it occurs in cardiac strain, is fully discussed under that subject.

Symptoms.—When this condition is fully developed, the condition of the circulation is one of compensation, closely resembling that met with in pure hypertrophy. There are no symptoms directly referable to it, and the subject of this condition is able to live a fairly active and comfortable life; but pallor or occasional faintness in one set of cases, lividity and dyspnœa in another, represent the effects of the original valvular defect. In other words, the clinical evidences of dilatation with efficient hypertrophy are not the existence but the removal or prevention of cardiac disorders of different kinds, particularly palpitation, pain, dyspnœa, and a disposition to syncope. At the same time such symptoms make their appearance with less provocation than in simple hypertrophy, the efficiency of the heart breaking down, and the form of enlargement to be next described—dilatation with failure—being induced for a shorter or longer period of time.

Physical signs.—The physical signs of dilatation with efficient hypertrophy are best seen in connection with the left ventricle in aortic incompetence. They consist in marked bulging of the præcordia; an extensive, powerful, heaving impulse; displacement of the apex-beat downwards and outwards, perhaps as far as the sixth or seventh interspace; increase of transverse percussion dulness to the left, and to the right also, if the right ventricle be simultaneously enlarged; and moderate increase of the vertical dulness along the left border of the sternum. The most striking auscultatory signs are the associated murmurs, not uncommonly accompanied with perceptible agitation of the stethoscope and the head of the observer.

Course.—The course of dilatation with efficient hypertrophy is similar to that of pure hypertrophy. It occupies several stages—the establishment of compensation, the maintenance of compensation, failure, and restoration of compensation—at least for a time. The termination is usually by gradual failure; occasionally by paralysis of the myocardium and sudden death. The duration is very variable in different instances, from months to many years.

Diagnosis and prognosis.—The same difficulties attend the diagnosis of dilatation of the heart with efficient hypertrophy as we have already studied in discussing hypertrophy; certain morbid conditions simulate the existence of cardiac enlargement, others conceal or obscure it.

From simple hypertrophy, say of the left ventricle, concomitant dilatation is diagnosed by greater bulging of the præcordia; by the more diffused and heaving character of the præcordial impulse; by greater displacement of the apex-beat towards the left as well as downwards; and by the greater increase of the transverse dulness in the same direction. The auscultatory signs are characteristic of the valvular lesion, not of the enlargement. The diagnosis from dilatation with failure will be discussed under that head.

The prognosis of dilatation with efficient hypertrophy is less favourable than that of simple hypertrophy. The condition of the patient as a whole is more precarious, an increased burden and expenditure of energy being entailed on the heart. Otherwise the prospect of life is subject to the same considerations as we have already discussed. A large number of circumstances have to be taken into account before the prospect of maintaining compensation can be correctly estimated in any instance.

Treatment.—In this form of enlargement of the heart the dilatation is a permanent result of damage of the valves, and has to be accepted as beyond remedy. In other words, the dilatation cannot be reduced unless the valve be rendered again competent. The leading therapeutical indication is therefore to maintain the efficiency of the hypertrophy. How this may be accomplished is fully discussed under its own head.

DILATATION WITH FAILURE.

Syn., Dilatation from Insufficient Emptying.

This is an increase in the capacity of one or more of the cardiac chambers, associated with inefficiency (absolute or relative) of the walls. There may or may not be accompanying hypertrophy, and also dilatation from overfilling.

Etiology.—Dilatation with failure of the heart occurs in two different connections: primarily, when it involves a previously sound organ; secondarily, when it supervenes on preceding enlargement—whether pure hypertrophy, or dilatation with compensatory hypertrophy.

Primary or simple dilatation of the heart is comparatively uncommon. It is met with in acute specific febrile diseases, particularly scarlet fever, diphtheria, and typhoid; in acute nephritis; as an effect of certain poisons, such as alcohol, tobacco, and digitalis; and in association with severe anæmia. It is also a result of violent muscular efforts, that is, acute strain.

Secondary dilatation of the heart with failure is very common, being the condition familiarly associated with the oncome of dropsy and other phenomena in valvular disease, and in failure of the heart in chronic nephritis. Under these circumstances, dilatation usually can be traced to one or more of the following causes—(1) disease or malnutrition of the myocardium, whether myocarditis or the result of coronary disease, general anæmia (however induced), disorders of assimilation, metabolism, and elimination (sometimes from insufficient exercise), indigestion and defects of diet (including over- as well as under-feeding); (2) chronic cardiac poisons, especially alcohol, tobacco, tea, the abuse of drugs, and retained excreta (uric acid and urea); (3) nervous influences, depressing or exhausting the cardiac ganglia, directly or reflexly; (4) acute disease, variously disturbing the heart, such as rheumatism, influenza, bronchitis, and typhoid fever; (5) muscular exertion or overwork, one of the commonest causes of all; and (6) fresh valvular lesions, or slow increase of the lesion to an extreme (unreasonable) degree, as in some cases of mitral stenosis. It most frequently happens that (7) combinations of these causes are at work; for instance, in the poorer subjects of valvular disease, and in the female sex. We must also observe that sooner or later, according to its form and severity, chronic valvular disease with compensation itself disposes to failure, by (8) establishing a vicious circle of slow, progressive impairment of the viscera and their great vital functions—the lungs, liver, stomach, bowels, kidneys, indeed the myocardium itself.

Morbid anatomy.—In primary or simple dilatation the weight of the heart is not increased, notwithstanding the visible enlargement, which proves to be due to increased capacity of one or more of the cavities with thinning of the walls, the left ventricle being the chief seat of the change. The muscular tissue is pale, soft, and flaccid; and is found on microscopical examination to be granular, fatty, or the seat of inflammatory and hæmorrhagic changes.

In dilatation with failure of a heart which had previously been the seat of hypertrophy or of dilatation with efficient hypertrophy, or in which permanent damage had been done by acute strain, the two forms of enlargement are found associated in various degrees, and the weight and appearance vary accordingly. The myocardium is usually very red and firm (cyanotic induration), from long-standing mechanical congestion and fibroid change; but granular or fatty degeneration often coexists. Mechanical congestion and cyanotic induration are found in connection with the other viscera; and the serous spaces and sacs are the seats of dropsy.

Pathology.—Dilatation of the heart with failure originates in insufficient emptying of the cardiac chambers. All the phenomena point to incompetence of the heart. Whether from defective or disordered nutrition of the wall, from abnormal peripheral resistance, from excessive amount of blood received and to be discharged, or from combinations of these causes, as we have seen under the head of Etiology, the heart fails —fails to complete its evacuation: the end of systole finds a proportion of the contents left undischarged. According to some authorities, the ventricles are never completely emptied at the end of systole. The truth is that the capacity of the cardiac chambers—that is, the volume of their contents at the end of systole and diastole respectively—is not rigidly fixed, as we are accustomed to regard it in our discussions of the mechanism of the heart in health and disease. The high extensibility of their walls provides for considerable variations in the amount of influx, and in the degree of resistance, and in the relation of these to each other. The argument is not affected by this consideration. On the top of this residue the chamber receives its ordinary charge. Its walls are stretched till they provide accommodation for the increased amount of blood; it is over-charged, over-distended, dilated. This dilatation is different in kind from the dilatation previously studied—dilatation from over-filling. It is dilatation from insufficient emptying—residual dilatation. It is a result of deficiency, not of excess; of parietal, not valvular, incompetence. It originates in physiological, not physical, failure of mechanism: in failure of the muscular walls of the heart to display and maintain a sufficient amount of force. If it continue it increases in degree, strains the muscular walls, spreads backwards in the form of mechanical congestion of the viscera and dropsy, and relieves itself spontaneously by hæmorrhage and fluxes. In very rapid cases it may prove fatal by sudden spasm or paralysis of the over-distended cardiac chamber, or by actual rupture of the heart. In one respect, however, it is more favourable than the other kind: it is often remediable, because it is after all but an accessory misfortune, which might be removed by attention to its cause and to the driving power of the heart.

In other instances of dilatation, the fibres are over-stretched by over-distension during violent or sustained muscular efforts; and they remain so, the dilatation being followed by hypertrophy. Happily the extensibility of the cardiac and venous walls much more frequently bears the strain, at least in young subjects, and permits temporary dilatation during violent efforts, at the cost of nothing more than embarrassment, until the chamber is relieved both forwards through the arteries and backwards through the veins (safety valve action). Indeed, not only in these but in all cases, dilatation might be regarded as a desperate means of temporary relief, that is, of averting even more serious consequences; and in ordinary cardiac failure associated with valvular disease, or chronic nephritis, spontaneous recovery often takes place when the patient is compelled to rest, or with the occurrence of hæmorrhage, dropsy, and fluxes. Still, dilatation with failure is a highly dangerous, distressing, and uncertain phase of cardiac disease, and one which calls urgently for the assistance of art. The balance between work and force, between load to be driven and driving power, between circulatory obstruction and cardiac strength, is disturbed. The symptoms of heart disease are the phenomena of dilatation with failure; its signs are the phenomena of increased size with weakness of the heart.

Symptoms.—**General.**—The clinical phenomena of dilatation with failure of the heart in its ordinary form are familiar to us under the name of cardiac dropsy. When they are fully developed in a severe case, the patient is confined to bed, or to a chair, in a sitting posture, suffering from orthopnœa. His countenance is anxious, full, and anæmic, with a tint of lividity and jaundice. The integuments of the lower extremities and lower part of the body are dropsical, the exact distribution of the fluid depending on the posture maintained, and changing with it. The upper part of the trunk, neck, and face are less dropsical, but definitely puffy. Dyspnœa, even in the sitting posture, may be an urgent symptom, with occasional cough and the expectoration of a frothy fluid, possibly stained with blood. Complaint is made not only of these symptoms, but of local cardiac distress—a sense of oppression or actual pain in the præcordia, palpitation, and faintness. Sleep is impossible, or comes in short broken snatches, from which the patient awakes in alarm. In other instances, a dull, heavy, somnolent condition continues for days and nights, in association with lividity and semi-asphyxia. The appetite is impaired, yet light solids can usually be taken without discomfort and with benefit. Flatulence is often urgent, and greatly aggravates cardiac distress. Vomiting may occur, with the rejection of food and mucus, possibly blood-stained. The bowels may be either confined or much relaxed, constituting intestinal flux. The urine is scanty—falling to 30, 20, 10 fluid oz., or even less; it is high-coloured and very acid, deposits lithates, and contains albumin and sometimes hyaline casts.

It is scarcely necessary to add that all these distressing phenomena and disturbances are not present in every case of dilatation of the heart with failure. The clinical characters of acute dilatation of the heart, as it occurs in scarlet fever and diphtheria, are described under "Acute Myocarditis"; those of acute dilatation, from sudden or severe muscular exertion, in the section on "Strain."

Physical signs.—The physical signs of dilatation with failure of the heart are different according as the condition is primary or secondary; and when secondary, according to the form and cause of the cardiac enlargement on which it supervenes. Only a general description, therefore, will be given (see also section on Strain). The præcordial impulse is weak and diffused. The apex-beat is situated below and to the left of the normal, often far beyond the nipple line, and is usually feeble—always more feeble than before in the case of the failing heart of valvular disease. It is often irregular in force and rhythm; in advanced cases there is continuous or occasional asystolism. Transverse præcordial dulness is increased far to the left; and usually well beyond the right border of the sternum, or even half-way to the right nipple line. Vertical dulness is sometimes found increased; possibly, however, the normal limit upwards, at the fourth left costal cartilage, may not be perceptibly exceeded. The exact dimensions and outline of the area of dulness naturally vary with the primary disease. The auscultatory signs are also necessarily various. The first sound becomes short and sharp, like the second, in primary dilatation. In the failing heart of chronic Bright's disease, where the process of dilatation can be carefully observed, the characteristic *bruit de galop*, or cantering rhythm, is developed, and may continue appreciable for weeks or months. Sometimes a systolic murmur appears at the mitral apex, significant of dilatation of the mitral orifice, *i.e.* leakage through the mitral valve. The same phenomenon occurs at the right auriculo-ventricular orifice in failure of the right ventricle, significant of tricuspid regurgitation. On the other hand, previous murmurs of structural origin may disappear with the supervention of dilatation, from sheer failure of the heart to raise a *bruit*. Cardiac failure in mitral obstruction illustrates both of these effects on the auscultatory signs; the presystolic murmur disappears (perhaps temporarily), and a tricuspid systolic murmur replaces it.

The cervical veins become distended, pulsate, and may even fill from below. Dulness and abundant small moist râles are found over the bases of the lungs; in severe, and especially in acute, cases râles may be almost universal, with urgent dyspnœa and hæmoptysis. Very often there are signs of hydrothorax on one or both sides. The liver is found to be large and tender, and occasionally it pulsates. Ascites is common.

The pulse varies greatly, according to the initial disease; but it may be said to lose force and volume as dilatation progresses, whilst it becomes more frequent and irregular.

Course.—The symptoms and signs just described may either last for a short time, whereupon they decline and disappear, or become steadily aggravated until death occurs. It is unnecessary in this place to enter further into details. When dilatation with failure passes off in secondary cases, we say that compensation is restored; but sooner or later, under the influence of the same or other causes, hypertrophy fails again, dilatation from insufficient emptying returns, and all the phenomena reappear. The clinical history of some cardiac patients consists of alternate periods of these two conditions. In primary dilatation, *e.g.* the acute dilatation of scarlet fever, the heart is believed to recover its ordinary size if the patient recover.

Diagnosis.—Two important steps have to be taken in the diagnosis of dilatation of the heart with failure. The condition is diagnosed, in the first place, from the two other kinds of enlargement; that is, we determine the essential fact which lies at the foundation of the prognosis and treatment of cardiac disease—the existence of compensaton or of failure.

This part of the diagnosis is to be made by attentive observation and consideration of the symptoms and physical signs. Cardiac distress, dropsy, albuminuria, and the other phenomena of mechanical congestion, at once suggest failure; and this is confirmed by the discovery of the large tender liver, the distended veins in the neck, the appearance of the face, the signs of enlargement of the right ventricle, of tension in the pulmonary artery, of œdema at the bases of the lungs. On the contrary, compensation, whether by pure hypertrophy or by dilatation with efficient hypertrophy, is evidenced by the absence of all symptoms of distress and arrears of work.

The chief difficulty that is felt in diagnosis arises in connection with cases accompanied by slight or few symptoms: breathlessness and cough only on exertion, occasional faintness, palpitation, oppression or pain in the præcordia, with suspicious fulness of the ankles. Is this failure, or is it compensation? A study of this class of cases enables us to appreciate more clearly the nature and meaning of compensation. In them compensation is precarious: the balance of the pressure is shifting from the one side to the other, now for, now against, the heart, in exact correspondence with the passing influences upon it—a muscular effort, anxiety, indigestion, weather, the activity of elimination, and the like. The condition is one of unstable equilibrium, and demands attention, as being unsound, for the only condition of an active organ that can be regarded as satisfactory and therefore normal is stability of function, based upon a considerable reserve of energy. Conversely stated, there must naturally be a number of cases, and there must naturally be appreciable periods or phases in the history of every case of enlargement of the heart, in which it is difficult to say whether the condition for the time being is one of compensation or one of failure: first, at the establishment of compensation; secondly, when it is taxed and threatening to break down; thirdly, when it is being restored. The recognition of the kind of enlargement ought therefore to be carried so far as to determine whether the condition is one of imperfectly established compensation, perfect compensation, failing compensation, or failure.

The second step in the diagnosis is too often neglected: determination of the cause of the failure. Before we can forecast the issue of failure of the heart, or plan recovery from it, we must make ourselves acquainted with its *origin*, by careful inquiry in each individual instance whether over-work, poverty, anxiety, alcohol, gout, pulmonary disease, etc., has been the cause, as set forth under the head of Etiology.

Prognosis.—In acute primary dilatation with failure the prognosis is very grave, but not hopeless.

The prospect of recovery from cardiac dropsy depends on the circumstances with which it is associated in different instances. These must always be ascertained and duly weighed, for instance (1) the primary lesion—whether valvular (and if so, its situation), pericardial, pulmonary, or renal; and (2) the cause of failure, as detailed in the paragraph on Etiology. When the particular cause at work, such as over-work or malnutrition, can be removed by ordinary treatment, the prognosis is relatively good. When the cause cannot be influenced, the prospect is unpromising, *e.g.* when the coronaries are degenerated, the kidneys diseased, the nervous conditions unfavourable, or the limit of compensation reached, in consequence of secondary changes in the abdominal viscera, lungs, and myocardium. The prognosis of cardiac failure is essentially different at every age of life.

Treatment.—The principal indications for treatment in dilatation with failure of the heart are four—(1) Attention to the cause of failure; (2) sufficient evacuation of the cardiac chambers and mechanically congested vessels and viscera; (3) increase of the cardiac force; and (4) relief of distress and disorder.

The cause of failure is to be removed, reduced, counteracted, or otherwise combated, as far as possible, and as speedily as possible. Failure is ordinarily due, not to something within the heart and inaccessible, but to something outside the heart and capable of control. The over-worked labourer is put to bed, the ill-nourished subject is properly fed, the poor man is sent into a comfortable hospital, the anæmic girl is given aperients and iron, the over-fed and coddled boy has his diet reformed and commences graduated exercises in the open air. Alcohol and tobacco are interdicted. These are instances of the application of this elementary principle of treatment which must never be neglected. Unfortunately the cause is sometimes undiscoverable; and still more frequently it is intractable, such as coronary degeneration, Bright's disease, progressive lesions, and nervous depression.

Sufficient emptying of the cardiac chambers and veins is to be effected by removing residues from behind and reducing the resistance ahead. In very urgent cases—primary acute dilatation in scarlet fever, etc.—abstraction of blood by means of leeches, cupping, or venesection should be practised; and the same means are successful in urgent failure of the right heart in mitral disease and emphysema. More commonly, that is in ordinary cases of cardiac dropsy, we are content to reduce the venous tension by brisk saline and other hydragogue purgatives, particularly compound jalap powder. These afford remarkable relief of the mechanical difficulty. When these fail, or when the condition of failure is more advanced and extensive, paracentesis of the serous cavities and puncture of the subcutaneous spaces, *secundum artem*, must be carried out. In every instance bodily rest is ordered, which reduces peripheral resistance and contributes to the relief of the heart *a fronte*. Purgation, iodide of potassium and the nitrites, have also this effect.

Increase of the cardiac force is evoked by means of food and nervo-muscular stimulants. Food must be nutritious, and therewith easily digestible and non-flatulent. Light solids should be ordered, unless distinctly contra-indicated, including small quantities of lean mutton, fowl, fish, and bread. A liquid diet is, as a rule, to be avoided; but if sickness, nausea, or complete anorexia be present, as is not uncommon in the acute phases of cardiac failure, recourse must be had to beef essences, milk, or koumiss, peptonised foods, and the like. Occasionally it is necessary to remove all food for a day, and give only small doses of alcohol in water.

In less urgent cases a moderate allowance of pure alcoholic stimulant, such as brandy or wine, to be taken either at ordinary meal times or with an "extra" between these, is usually beneficial. This reference to alcohol introduces us to the use of cardiac stimulants. In acute failure of the heart, brandy is the most readily available remedy, given by the mouth or rectum. Ether or strychnine subcutaneously is even more swift and powerful, if at hand. Short of this degree of urgency, we employ the ordinary *mistura stimulans*, consisting of ether, ammonia, and aromatics.

All these remedies are of swift and usually temporary action on the heart. In the digitalis group we have a series of drugs which act less quickly and less certainly, but much more persistently, upon the circulation. Digitalis, strophanthus, squill, senega, caffeine, cactus, and convallaria have the power of evoking more force from the labouring cardiac wall, slowing the rhythm—thus resting the ventricles and draining the veins, increasing the arterial tension generally—whilst presently lowering it in the renal vessels, and finally effecting diuresis, which is both the means and the measure of the restoration of the circulation. The details of the selection and administration of these drugs, depending on the nature of the primary lesion and other circumstances, are discussed in the chapter on "Valvular Disease." The point to be observed here is, that the leading indication for their use is not this or that form of valvular disease, nor valvular disease at all, but dilatation with failure of the heart.

With increased cardiac action and emptying of the over-distended veins and chambers, efficient hypertrophy begins to return. To restore it perfectly, all the measures just reviewed must be continued for a sufficient length of time—the cardiac stimulants, now given in smaller or tonic doses, careful feeding, and prevention of over-distension by regulation of the bowels. At this stage of recovery from dilatation there arises the question of exercise of the heart, in place of the comparative rest which had been enjoined. It is the same question essentially which arises during convalescence from disease of every kind—When is rest to be interrupted and work resumed in a safe and beneficial form?—remembering also that the heart is an organ peculiarly intended to work. The practical answer is given by making a trial. The patient is allowed to sit up, to stand, to walk; and the effects are carefully observed in the pulse, breathing, subjective sensations, and return or absence of œdema. All going well, an advance is made daily in a gradual manner, until the patient after a time is able to return to comparative activity. Instead of these somewhat ill-defined forms and stages of work, several systems of graduated cardiac exercises have lately been introduced for the purpose of improving the muscular vigour, tone, condition, or form of the heart. The chief of these are Oertel's system of graduated hill-climbing and Schott's Nauheim system of resistance movements (combined with carbonic acid baths). These systems are different only in form from the regulated out-door exercise which has always been recommended in the treatment of chronic cardiac disease in this country since the days of Stokes.

Whilst the preceding indications are being attended to, there is urgent necessity for the relief of distress. If the pathological significance of dyspnœa, pain, hæmoptysis, palpitation, faintness, insomnia, and the other symptoms of cardiac failure be intelligently understood by the practitioner, the rational remedies for relief will readily occur to his mind. Indeed, attention to the foregoing principles will usually have this effect of itself; that is, by means of rest, proper feeding, evacuation of the bowels, and well-selected drugs. Urgent præcordial distress demands ether, ammonia, alcohol, and other stimulants and carminatives, particular attention being directed to the stomach, and the diet reconsidered. Severe pain, especially of an anginal type, indicates the use of nitrites. Extreme dyspnœa and failure of pulse are speedily relieved by strychnine subcutaneously. Dyspnœa with great lividity and hæmoptysis should be treated with free leeching over the sternum, cupping between the shoulders, or venesection, as we have already seen, rather than with "symptomatic" drugs. A simple carminative mixture should always be at hand to effect removal of flatulence: but here again it is better to avoid and prevent distress, by strict dieting and occasional doses of calomel, than to trust to removing it.

NEW GROWTHS AND PARASITES.

New growths in connection with the heart are a subject of pathological rather than of clinical interest. The myocardium, endocardium, and pericardium are occasionally the seats of carcinoma, sarcoma, lymphoma, myoma, lipoma, and fibroma. Malignant disease is usually secondary to growths in the neighbouring structures, such as the mediastinal glands, lungs, or œsophagus. Scrofulous masses, miliary tuberculosis, and actinomycosis are also met with in the heart; and so are the echinococcus and cysticercus cellulosæ. Syphilitic lesions of the heart are described separately. It cannot be said that new growths or parasites in the heart give rise to phenomena, or run a clinical course, sufficiently distinctive to justify a diagnosis of their presence. Where cardiac distress is a prominent symptom in patients suffering from malignant disease, it would naturally suggest invasion of the heart; but it might be due to involvement of the neighbouring viscera and nerve plexuses.

The prognosis is hopeless; the treatment palliative only.

SYPHILIS.

Two well-marked forms of syphilitic lesion are found in connection with the heart.

1. Gummata.—These present considerable variety in number, size, and age. According to their situation and other circumstances, they are associated with fibrosis of the interstitial tissues (chronic myocarditis), chronic endocarditis, chronic pericarditis, cicatricial puckering of the walls, and cardiac aneurysm.

2. Syphilis of the Coronary Arteries.—This may lead to obstruction of an arterial branch, and thus either to infarction of the myocardium or development of a fibroid patch (see "Chronic Myocarditis"), or to fatty degeneration.

Symptoms and diagnosis.—Syphilis of the heart has frequently been found post-mortem in the bodies of persons who had presented neither symptoms nor signs of cardiac disease. In a considerable number of instances, however, disturbance of the frequency and rhythm of the heart and pulse has been a prominent feature. In other cases, pain and different forms of præcordial distress have been complained of; and the physical signs observed have included those of enlargement and pericardial and endocardial murmurs. Sudden death has been relatively common; a circumstance which has afforded unusual opportunities of studying the disease post-mortem.

Syphilis of the heart might be more frequently diagnosed than it is at present, if the relation of the coronary arteries to cardiac nutrition and action were carefully kept before the practitioner's mind. Failure of the heart, whether in chronic valvular disease or independently of it (idiopathic enlargement), occurring in a syphilitic subject, without other more ordinary cause to be found for it, such as over-work or privation, might reasonably be referred to obliterative narrowing of a branch or trunk of the coronary vessels. Further, if in such a case ordinary treatment with rest, diet, and cardiac tonics failed, with rapid increase of distress, whilst the substitution of iodide of potassium afforded relief, the diagnosis would be practically established.

Prognosis and treatment.—These subjects have been sufficiently indicated under the preceding heads.

J. MITCHELL BRUCE.

CONGENITAL HEART DISEASE.

Congenital affections of the heart are of more scientific interest than practical importance. They afford unlimited scope for speculation as regards their causation, and provide many difficulties in respect of diagnosis, yet they yield but little opportunity for successful treatment.

Etiology.—It is vain at present to discuss whether congenital heart lesions are most frequently produced by some inherent defect in development, or by some acquired disease during the antenatal period; it is customary to term causes belonging to the former group, *embryogenic*, and to the latter, *nosogenic*. No doubt both factors are at work in varying degrees. Diverse results, moreover, are probably produced by different degrees of resistance in the organism.

Congenital affections may be correlated with various phases of growth, and developmental processes seem to be interrupted at different periods. If arrest occurs at the stage when there is an auricle, a ventricle, and an aortic bulb, one of the rarer anomalies may result—a heart consisting only of an auricle and a ventricle. If interference occurs during the growth of the interventricular septum, an aperture is left in the undefended space. Similarly, when alterations occur in the growth of the auricular septum, the foramen ovale remains permanently open. When modifications occur in the evolution of the great arterial trunks, there may be several changes in their arrangement. Over and above such developmental lesions, structural alterations may be caused by intra-uterine disease. They are commonly the result of infection. It is undeniable that the endocardium is peculiarly liable to antenatal infection, which affects, by preference, the right side of the heart, probably from the relatively higher pressure as compared with the left side. According to the statistics of von Dusch, males are more liable than females to congenital heart affections, the proportion which he found being 64·6 of the former, and 25·4 per cent. of the latter.

Morbid anatomy.—The classification of congenital lesions of the heart is somewhat difficult, but the most satisfactory method consists in following the different anomalies from the simpler to the more complex.

Displacements.—Changes in the position of the heart occur in different directions. Amongst these alterations may be mentioned transposition, commonly associated with a similar change in the position of the other viscera. The heart, however, may be otherwise displaced, so as to occupy a position in the abdomen, or in the neck, or to lie external to the chest wall in the thoracic region. Transposition of the great arterial trunks is not infrequent, in which case the aorta rises from the right ventricle, and the pulmonary artery from the left. This condition is usually accompanied by other morbid changes.

Defects.—A deficiency of some part of the heart is a common cause of malformation. The entire heart may be absent, the pericardium may be wanting, or the internal septum may be defective. In this latter case the heart may only consist of three, or even of only two, chambers. More commonly, however, there is an aperture through the septum, either at the level of the foramen ovale or of the undefended space.

Numerical changes.—Alterations in the number of the cusps are not uncommon, whether by excess or deficiency. Such changes are more common as regards the arterial than the venous valves.

Persistent fœtal structures.—Some of the fœtal channels between the great blood vessels, obliterated normally at birth, may remain permanently open. The most common abnormality of this kind is persistence of the arterial duct. This is not uncommon as an isolated lesion, but is more usually found in association with other changes.

Valvular lesions are of common occurrence, and furnish, as a rule, intrinsic evidence of endocarditic origin. The pulmonary cusps are especially liable to such changes. Other valvular lesions, although not so frequently met with, are far from rare.

Complex lesions.—Many of the lesions which have been referred to are often found in association. Perhaps the most frequent combination is to be found in pulmonary obstruction along with incomplete septum ventriculorum, attended not uncommonly, also, by persistence of the arterial duct. In congenital heart lesions, there are commonly malformations elsewhere in the body, the existence of which seems a strong argument in favour of the embryogenic origin of many malformations of the heart. Such attendant alterations may consist in modifications in the form, or alterations in the number, of internal organs, but there may also be structural defect in the external conformation of the body.

Post-mortem examination of patients dying of congenital heart lesions, may show abundant evidence of effects produced by disturbance of the circulation; on the other hand, the lesion may not have produced the slightest interference with the flow of blood, and no effects can therefore be expected. In the former case there is a cyanotic condition of the tissues, showing itself in the enlarged size and deep colour of the solid organs—liver, spleen, and kidneys, with catarrh of the mucous membranes, and sometimes evidence of transudation into the serous sacs, including the meninges. One interesting point lies in the fact that pulmonary tuberculosis is a frequent sequel to congenital heart disease. This is proved by Peacock's statistics, showing that out of fifty-six patients with different forms of congenital cardiac malformation, who survived the age of 8 years, 16·7 per cent. died of tuberculous lesions, while the deaths from consumption during the same period amounted only to 9·1 per cent. of the population at large.

Symptoms and physical signs.—Congenital diseases of the heart sometimes give no general symptoms; in fact, unless there is some definite disturbance of the equilibrium of the circulation, symptoms are absent.

When general symptoms are present, they are produced by interference with the balance of the circulation. Cyanosis, always increased on exertion, is a common feature, and it is accompanied by increase of the specific gravity of the blood, augmentation of the hæmoglobin, and multiplication of the corpuscles. The specific gravity may reach 1070: the hæmoglobin may be 160 per cent.; and the numbers of red and white corpuscles may be respectively above 9,000,000 and 16,000 per cm. A tendency to hæmorrhage and transudation is associated with cyanosis, while the fingers and toes frequently show the well-known clubbing and arching. Breathlessness is present; it usually occurs on exertion, but sometimes appears also in a paroxysmal form, like cardiac asthma. Chilliness is commonly complained of, although the temperature may be normal, and a lethargic or apathetic condition is often observed. Convulsive attacks may be induced by exertion or coughing. A stunted condition is often seen, and other malformations are frequently associated with the cardiac lesion. Amongst local appearances there is sometimes an arching forward of the præcordia, no doubt due, for the most part, to enlargement of the right side of the heart.

The physical signs of congenital lesions are very various and often indefinite, unless clear evidence is present of some of the simpler changes, such as complete transposition of the heart or one of the valvular lesions. It will be advantageous to summarise the principal features of some of the commoner lesions.

Pulmonary orifice.—In obstruction, there is cyanosis with breathlessness, clubbing of the digits, and arching of the nails. The pulse is small and empty, the præcordia usually prominent. A thrill may be felt at the base with dulness increased to the right, and a systolic murmur in the second left intercostal space, often widely propagated, followed by a weak second sound. Incompetence is much less common. It presents, generally, symptoms closely similar to those just mentioned, with a diastolic murmur in the second left intercostal space carried down the left edge of the sternum, sometimes accompanied by a thrill.

Tricuspid orifice.—Affections of this are less common than of the pulmonary orifice. When present, they produce less cyanosis and dyspnœa, while clubbing of the fingers is generally absent. The pulse is not characteristic, but movements or turgidity of the cervical veins, epigastric pulsation, enlargement of cardiac dulness to the right, and presystolic or systolic murmurs, according to the lesion, may be present, the former sometimes with a corresponding thrill.

Incomplete auricular septum.—This, one of the commonest congenital lesions, rarely produces general symptoms or physical signs, when present as an isolated affection. Indeed, it may be said in many cases, when associated with other lesions, to prevent the development of such characteristic appearances as the other lesions might be expected to produce. It is easy to understand why there should be no characteristic effects produced by the lesion itself, seeing that the pressure in the auricles under ordinary circumstances is nearly equal. In one interesting case mentioned by Eichhorst—mitral disease with this defect—beautiful venous pulsation was seen in the neck.

Incomplete ventricular septum.—Much more definite symptoms result from defects in the septum of the ventricles than is the case in that of the auricles. The symptoms closely resemble those produced by mitral incompetence, with one essential difference—the lungs escape the effects of backward pressure to a considerable extent. A simple communication between the two ventricles sometimes produces a systolic murmur, on account of blood streaming from the left into the right ventricle. In a very large proportion of cases this defect is associated with other lesions, and its clinical results may be entirely overshadowed by the effects of the accompanying conditions.

Persistent arterial duct.—Patent ductus arteriosus is considered to be rare as an isolated lesion, yet five cases have been under my observation during the last three or four years in which this diagnosis was made, and in one of these it was verified after death. It is common in association with other congenital lesions. There is but little cyanosis or dyspnœa. There may be slight bulging of the second left intercostal space, and a late systolic thrill is often felt over this spot. The heart may be slightly enlarged on percussion, but the most characteristic feature is a murmur in the second or third left intercostal space; this murmur distinctly follows the first, and is continued beyond the second sound, which is frequently accentuated or reduplicated in the pulmonary area. When the duct is aneurysmal as well as patent, it may, by pressure upon the left recurrent laryngeal nerve, produce paralysis of the left vocal cord. An interesting observation was made by François-Franck: During deep inspiration in this condition, the radial pulse becomes smaller, on account of the expansion of the lungs, aspiration of blood from the pulmonary artery, and consequent increase of the stream from the aorta.

Diagnosis.—It is not, in general, difficult to determine that a congenital heart affection is present, but in many instances it is impossible to be absolutely certain whether lesions which may be present are really of congenital origin. The differential diagnosis of congenital lesions must be conducted in accordance with the general principles laid down in those sections dealing with acquired heart disease.

Prognosis.—The future in any case of congenital heart disease depends upon the nature of the lesion which is present, and also upon the general nutritive possibilities. Such lesions may not be attended by any considerable disturbance, and life in such instances is known to have been prolonged until the age of 60 years has been reached. If there be much disturbance, however, the results are otherwise. Defects in the septa only cause interference when the openings are large. Patent arterial duct in itself causes but little disturbance. Transposition of the main arteries renders the maintenance of life impossible. The prognosis will be formed in accordance with the presence or absence of symptoms of disturbance. If there be much dyspnœa, cyanosis, or hæmorrhage, the outlook is grave: and if, along with such symptoms, there should be complications, such as pulmonary collapse, capillary bronchitis, broncho-pneumonia, or tuberculosis of the lungs, the prospect is rendered still darker.

Treatment.—In those cases which present little or no disturbance, there will not be much difficulty in regard to management. Even in such cases, however, when possible, the patient's surroundings should be so arranged as to secure the most favourable conditions. A warm, dry climate, in which abundant fresh air and sunlight can be ensured, without undue exercise, will be found of paramount importance. The food must be light yet nutritious, and the clothing, while not heavy, must be sufficiently warm. It has been generally recognised, since the observations of Gintrac, that alcohol is prejudicial when cyanosis is present, but some cases I have met with in recent years, particularly one seen lately with one of my friends, have led me to suspect that this belief is not founded on very sure grounds. Certainly the employment of alcohol in some cases is urgently indicated. The treatment of the various symptoms which arise must be carried out in the manner described in the sections dealing with acquired lesions.

G. A. GIBSON.

NERVOUS DISEASES OF THE HEART.

INTRODUCTION.

In order to explain more easily the nature of nervous diseases of the heart, it may be advisable to commence by a short account of its nervous system. It was long ago shown by Harvey, that when the heart was cut into small pieces, each piece was capable of independent pulsation. The researches of Bowditch, Gaskell, and others have shown that such independent pulsation may occur even in parts of the heart in which neither ganglia nor nerves are discoverable. Rhythmical pulsation is therefore regarded at present as a property of the muscular tissue itself. Its amount and character depend on the condition of the muscle, and upon the various stimuli, mechanical or chemical, which act upon it. The co-ordination of the muscular contraction in each cavity of the heart, whether it be auricle or ventricle, depends partly upon the condition of the muscular tissue through which stimuli are propagated from one part to another, and probably also in large measure to the ganglia and fibres which are present in the organ.

One very instructive experiment in regard to these points is the application of a clamp to the middle of the frog's ventricle. When this is done, it has been found that the apex of the heart, which contains no nerves, usually stops; but if the intraventricular pressure be raised, the apex also will begin to beat, although at a different rhythm from the base, the stimulus of the increased pressure having brought about contraction in the muscular fibre of the apex itself. Local irritation in the ventricle of the frog's heart produces local weakness, so that if small points are injured by a pair of forceps, these points remain at the next ventricular contraction as small red projections upon the surface of the ventricle, the injured muscular tissue not having contracted at the same time as the rest. When the heart is dying, the muscle of the ventricle, instead of contracting all at once, does so in a somewhat peristaltic manner, so that the blood, instead of being driven out of the ventricle, may be only driven about hither and thither in the cavity.

The most remarkable alteration in the heart is perhaps that which ensues upon irritation of a point in the ventricular septum at the junction of the lower two-thirds with the upper third. It has been discovered by Kronecker that when this point is irritated by puncture or scratching with a needle, the heart immediately stands still, and its regular pulsations are replaced by a simple flickering movement of the muscular fibre.

Heat and cold have a powerful action upon the heart. When the isolated ventricle of the frog's heart is heated, it first of all begins to contract more quickly and more powerfully; its pulsations become more and more frequent, until at last it stands still in what is known as heat-tetanus. If it be quickly cooled down, the same phenomena occur in reverse order. The stand-still from heat is succeeded by frequent pulsations, which become gradually rarer, until they reach the normal; and if the cooling be carried still further, they become less and less frequent, until at last the heart stands still in complete diastole. Should the heart be heated beyond the point at which heat-tetanus occurs, it passes into a state of rigor, from which it is impossible to recover it. The mammalian heart is affected by heat and cold in like manner even when it is still present in the body, so that a rise of temperature quickens its pulsations, and if it be too high will stop the heart altogether. Cold, on the other hand, slows the pulsations, and will even stop them if the temperature of the animal be sufficiently lowered.

Certain poisons, of which digitalis may be taken as an example, slow the action of the ventricle, and as a rule arrest it in firm systole in the frog. This is not a condition of paralysis, because when the ventricle is distended by pressure from within, the heart will again recommence its pulsations. In mammals the heart is likewise arrested, but very frequently it is arrested in diastole.

The co-ordination of the different cavities of the heart may be due in some measure to transmission of stimuli from muscular fibre to muscular fibre, but the muscular connection between the auricles and ventricles of the mammalian heart is very slight indeed: and the fact observed by Ludwig, that by dropping tincture of opium into the heart, the ordinary rhythm may be completely altered and the ventricles made to contract before the auricles, appears to show that the co-ordination between the cavities is chiefly carried on by means of ganglia and nerves.

In the ordinary heart, contraction begins at the vena cava, spreads thence to the auricle, and from there to the ventricle. In this process the stimulation of each part consecutively is probably of three kinds—(1) Propagation of stimulus through the muscle; (2) propagation of stimulus through the ganglia and nerves; (3) stimulation by the increased pressure of the blood driven into each cavity.

In the case of the frog's heart, it has been shown by Gaskell that compression between the auricle and ventricle will interrupt the rhythm, so that instead of each auricular systole being followed by a ventricular contraction, three or four auricular contractions may occur before the ventricle responds.

Increased pressure within the ventricle does not always produce the same result. In the heart of the tortoise it was observed by Marcy that the heart beat less frequently the greater the resistance it had to overcome; but in the heart of the frog the contrary seems to be the case, and the heart generally beats more frequently, as if it were in a hurry to empty itself and remove the irritation caused to it by the increased pressure upon its internal surface. This appears also to be the case in mammals. It is to be noted that this tendency to more rapid pulsation from increased intracardiac pressure is due to the local action of the pressure upon the heart, but, as we will subsequently see, increased pressure within the arterial system usually has a totally different action, and tends to slow the heart when its connections with the central nervous system are intact, because the effect of the increased arterial tension on the vagus nuclei more than compensates for the accelerating action of increased pressure on the heart itself.

Having now considered the co-ordination of the muscular fibres in the cavities of the heart, and the relation of these cavities to one another, we must now consider the co-ordination of the heart-beat in relation to the wants of the body.

It is necessary for its physiological activity that any organ when in full action should receive a larger supply of blood than when at rest. There is not enough blood in the body to supply all the organs at once, and the supply to active organs is kept up, to a certain degree, by contraction of the vessels going to other organs which are not in action at the same time, so that the blood supply to the latter is diminished and more blood is available for the former. But, in addition to this, an increased supply of blood to active organs is brought about by increased action of the heart, and during great activity, either of muscular or glandular tissues, the heart as a rule beats more frequently. This was noticed by Marey as an exception to his general law that the heart beat more frequently the less difficulty it had in emptying itself. He observed that during digestion, when the glandular structures of the stomach and intestines were in full action, the blood pressure was, upon the whole, higher, and yet at the same time the pulse rate was greater.

The nerve-centre for regulating the distribution of blood is situated in the medulla oblongata, and may be said to consist of two parts—(1) For the heart, and (2) for the vessels. Each of these is again subdivided, that for the heart into (*a*) an inhibitory and (*b*) accelerating centre, by which the cardiac pulsations are slowed or quickened respectively.

The regulatory mechanism of the vessels consists of two parts: one (*a*) by which they are contracted, another (*b*) by which they are dilated. The contracting or vasomotor centre is situated for the most part in the medulla oblongata, but extends to a certain extent into the spinal cord. The vaso-dilating mechanism is not so concentrated as the vaso-constricting, but appears to be distributed throughout the spinal cord. The inhibitory or slowing centre sends its impulses to the heart through the vagus, and may either slow the heart or reduce the strength of its contraction, or both. The accelerating centre sends its impulses to the heart partly through the vagus, but chiefly through the sympathetic. The vasomotor centre sends its impulses to the vessels through the anterior roots of the nerves in the thoracic and upper lumbar region, which pass into the sympathetic ganglia of these districts, and thence along the splanchnic and other sympathetic nerves to the abdominal viscera and limbs.

RELATIONSHIP OF THE CARDIAC AND VASCULAR CENTRES.

When the various parts of the body are at rest and no demand for extra blood is made, either by the muscles or by the glands, the vaso-constrictor centre comes into play, so that the vessels generally are contracted and the supply of blood to each part is therefore limited. It is obvious that if at such a time the heart were to contract frequently and forcibly, the blood pressure would be unduly raised, and injury might occur from the rupture of some vessel under the extra strain. Provision has been made for this by the fact that increased blood pressure acts as a stimulus to the inhibitory centre in the medulla, and thus slows the heart through the vagus, so that the blood pressure is kept at its normal level. On the other hand, when the vessels are dilated and blood is pouring rapidly through the arteries into the veins, the pressure would tend to become abnormally low were it not that the mere fall of pressure appears to lessen the inhibitory activity of the vagus, and probably to stimulate the accelerating centres as well. In consequence of this, the heart beats more quickly, and thus tends to bring up the pressure to the normal. It is probable, however, that the acceleration is not due merely to actual diminution of blood pressure, but that during muscular and glandular activity certain substances are formed which have a stimulating action on the accelerating centre; and thus, in some cases, the acceleration may last for a considerable period after the diminution of the blood pressure which has brought it about has ceased. It even may occur during digestion, when the blood pressure is above the normal.

These centres thus act, as a rule, co-ordinately, tending to maintain the blood pressure at a more or less constant level. They may be excited reflexly by stimulation of various organs. Thus the inhibitory, or vagus centre, as it is often called, may be stimulated reflexly by sensory impressions made upon the nose, larynx, stomach, intestines, and, indeed, sensory nerves generally. Such impressions usually stimulate the vaso-motor centre at the same time, and thus maintain or even raise the blood pressure in spite of the slow action of the heart; but, when they are excessive, they may lead to slow action of the heart along with great lowering of the blood pressure, as in cases of shock.

It is obvious that if the blood pressure should be increased very greatly, it might not only lead to danger from rupture of a vessel, but might seriously interfere with the power of the heart to empty itself, and in cases of fatty degeneration of the heart might stop it entirely.

It has already been mentioned that increased pressure within the heart itself seems to stimulate it to greater activity, but at the same time the rise of pressure in the vessels, acting on the medulla, slows the heart through the vagus, so that, as a general rule, increased pressure with intact vagus causes the heart to become slow. On account of the slowness of the heart the blood pressure tends to fall, and thus injury is prevented either to the vessels in the brain or other vascular districts, or to the heart itself. Moreover, nearly everything which weakens the heart tends to increase the action of the vagus upon it; so that when the heart is poisoned by salts of potassium and certain metals or alkaloids, the power of the vagus is increased, and a stimulus of a given strength applied to the vagus trunk will slow the heart more than it would under ordinary circumstances. This increased strength of the vagus in a weak heart thus tends to safeguard the organ from over-pressure.

It is possible that even with a very slowly-acting heart the pressure in the vessel might be too much for it, and therefore there are nerves, known as the depressor, which pass to the vasomotor centre and arrest its action, so that the vessels, chiefly those of the splanchnic area, become dilated, and the blood pressure falls considerably, so that the heart is relieved.

NERVOUS DISTURBANCES OF THE HEART.

With this short summary of the nervous mechanism of the heart and vessels, we may now proceed to consider the nervous disturbances of the heart. These may be divided into motor and sensory, and these again may be subdivided in the manner which I have indicated for clearness in tabular form :—

SENSORY AND MOTOR DISTURBANCES.

PALPITATION	Emotional.	
	Reflex	From stomach. From intestines. From liver and gall duct. From urinary passages. From genital organs.
TOXIC		Tea. Coffee. Tobacco.

SENSORY DISTURBANCES.

UNEASINESS	Emotional, toxic, or reflex causes.
WEIGHT	
OPPRESSION	
DISTENSION	
PAIN	Pericarditis, angina pectoris.
DYSPNŒA	Cardiac asthma.

MOTOR DISTURBANCES.

Rate.	FREQUENCY	From paralysis of inhibitory fibres of the vagi	by neuritis	in alcoholism. after diphtheria.
			by poison	belladonna. tobacco.
		Cause uncertain	Emotion. Exophthalmic goitre. Paroxysmal tachycardia. Angina pectoris occasionally.	
		From stimulation of accelerating nerves	Fever. Cerebral abscess.	
	INFREQUENCY	From weakness of cardiac muscle	Fatty degeneration of the heart. Fibroid ,, ,, ,, Weakness after fevers, especially infective fevers, *e.g.* influenza or typhoid. Weakness after childbirth.	
		Increased resistance	Aortic obstruction. High tension from gout. Emphysema	
		From poisons	Digitalis and its congeners. Tobacco. Aconite.	
		Reflex stimulation of inhibitory centres	From stomach and intestines, kidney, lung, liver.	Flatulence or distension. Inflammation of stomach or intestines (sometimes).
		Stimulation of inhibitory centres	Lesions of brain, medulla, and cervical cord, *e.g.* meningitis, apoplexy, tumour, increased intracranial pressure, and disseminated sclerosis. Some mental diseases.	

MOTOR DISTURBANCES—*continued.*

Rhythm.	IRREGULARITY .	*Pulsus paradoxus* (more frequent during inspiration than expiration)	in cardiac weakness. in chronic pericarditis. in the presence of fibrous bands around the root of the aorta.
		Intermittent (beat occasionally omitted)	in chronic gout. in dilatation of the heart. in fatty degeneration of the heart. in fibroid degeneration of the heart. in sclerosis of the coronary arteries.
		Irregular (strong and weak varieties, bigeminal and trigeminal)	in mitral disease. after administration of cardiac tonics, or abuse of tea, coffee, or tobacco. in chronic gout.
		Delirium cordis (so irregular as to be uncountable)	in dilatation.
		Fœtal type (pauses similar in length and sounds in character)	in the later stages of fevers in extreme dilatation.
		Gallop rhythm (rapid action with reduplication of second sound)	in interstitial nephritis. in atheroma.

CHANGES IN FORCE.

The strength of the heart is generally estimated either from the more or less forcible character of the apex-beat or from the pulse as felt in the arteries. Both of these may be changed by an alteration (*a*) in the actual strength of the cardiac contraction, and (*b*) in the amount of blood which is contained in the ventricle at the time of systole. It is by no means easy to differentiate between the operation of these two causes, and they very often act together. Thus it happens that when the pulse is irregular in time it is also unequal in force, because a prolonged diastole allows the ventricle to become full, while a short diastole tends to lessen the amount of blood which can pour into it from the auricle, and thus to render the quick beats small, while the slow beats are fuller and apparently stronger. In mitral stenosis, for example, the ventricular pulsations are often apparently unequal in strength, although the heart may not be enfeebled; and the inequality in strength is probably due simply to irregularity of the rhythm. In shock also, such as occurs from a blow upon the abdomen or perforation of the intestine, the small feeble pulse is probably due to emptiness of the arteries from stagnation of the blood in the abdominal veins, as well as to actual feebleness of the heart itself.

Pulsations of undue strength may occur from hypertrophy of the heart, a condition the symptoms of which are discussed in another article. They may occur also in consequence of over-exertion or from nervous influences, which will be discussed later on under the head of "Palpitation."

Weak beats of the heart may be due to feebleness of the cardiac muscular fibre, caused by the presence of inflammation in the pericardium, endocardium, or muscular structure itself. This may also be due to weakness of the muscular fibres from degeneration, either fatty or fibroid. Such degeneration may be consequent on high temperature occurring in acute fevers, or to chronic malnutrition in wasting diseases. Weakness of the pulsations may be caused also by the action of the inhibitory nervous apparatus on the heart, either in consequence of depressing emotions, such as sorrow, fear, or anxiety, or reflexly from severe injury, and especially from severe inflammation or injury to the abdominal organs, as by perforation of the stomach or intestines. It may be caused also by inanition, and by low temperature consequent upon exposure to cold or the action of certain drugs of an antipyretic nature, especially when these have reduced the temperature rapidly from high fever. Such drugs, and also chloral, aconite, and various other poisons, may weaken the heart independently of any action upon the temperature. An unequal pulse generally indicates weakness of the heart, except in mitral stenosis, as I have already mentioned.

CHANGES IN RATE.

The normal pulse rate varies considerably in different individuals. It is usually somewhere between 60 and 80. It is generally greater in women than in men, and also in small than in large persons. The vagus in a healthy man exercises a constant inhibitory influence upon the heart, and when this influence is removed the pulse becomes enormously increased. It is known from experiments upon animals that atropine completely paralyses the ends of the vagus in the heart, and we may assume that in poisoning by atropine or belladonna in man the same condition occurs. In such cases of poisoning the pulse rate in man is usually 120. This may be regarded as the rate at which the human heart will beat when the vagus is paralysed.

A pulse may be accelerated normally by exertion, and its frequency may, as already stated, continue after the exertion is over. During swallowing, as has been shown by Kronecker, the pulse may be very much accelerated; and from my own observation I have found it quickened from 68 to 92, but the rate of increase varies much in different individuals and according to the mode of swallowing.

Heat, whether externally applied, as in a hot bath, or internally, as by hot drinks, especially hot alcoholic drinks, or when generated in the body, as in fever, quickens the pulse. The ordinary rate of acceleration in fever is, according to Liebermeister, eight beats for every degree centrigrade of temperature, but this does not always hold. In many diseases one finds that the pulse rate is 120, but in typhoid it very often is 96 irrespective of the temperature, so that one gets the impression that in typhoid there is some substance circulating in the blood which prevents the acceleration of the pulse which the raised temperature would otherwise produce. In scarlet fever, on the other hand, the acceleration is even greater than one would expect from the temperature, as if a toxine having a quickening action were present in the circulation. In endocarditis the pulse is more frequent than the rise of temperature alone would account for. The acceleration in this case appears to be due to the local stimulation of the heart, caused by the inflammation.

Acceleration from emotion occurs very frequently, and should always be allowed for in examining patients. It may occur in patients whose look would not lead one to imagine that they were at all emotional; and in examining healthy men for life assurance, I have generally found a very much greater emotional acceleration of the pulse than amongst patients in ordinary practice.

TACHYCARDIA.

The physiological causes of a frequent pulse have been already discussed, but the heart's action may be permanently or intermittently rapid from morbid conditions.

Etiology.—Tachycardia, persisting for a longer or shorter time, has been observed after diphtheria, probably in consequence of neuritis of the vagus similar to that which occurs in other efferent nerves. I have observed a persistently high pulse rate also in cases of alcoholism; and as in one of those at least digitalis failed to produce any slowing of the pulse, I am inclined to attribute the tachycardia to alcoholic neuritis affecting the inhibitory fibres of the vagus. Tachycardia is also a marked symptom in exophthalmic goître. In the three conditions just mentioned, the pulse remains persistently frequent, usually from 100 to 120 or more, but its rate is capable of being increased by exertion or emotion.

Paroxysmal tachycardia occurs in fits, which come on at varying intervals and may last from a few minutes to some days. In fifty-three cases collected by Herringham, thirty were in men and twenty-three in women. Of four cases in which the ages were given, only two were children, but in five others the history dated from childhood. Of these seven cases, five were in females. In twelve out of fifty-three, the attacks began between 20 and 30 years of age; in thirteen between 30 and 40; in ten between 40 and 50; and in three over 50. In one case the disease appeared to be hereditary for four generations, and in another hereditary liability was probable. In some cases, rheumatism, valvular disease, and syphilis preceded the attacks, but a causal connection between them could not be definitely traced. A considerable portion of those cases in which a cause could be observed have followed sudden or severe exertion. Paroxysmal tachycardia has been noted in some women at the menopause, and in others suffering from ovarian or uterine irritation. It has been also noted by Eccles and others in cases of floating kidney, by Rosenbach in gastric disorders, and by Payne Cotton as the result of tapeworms. I have myself observed tachycardia accompanying an attack of angina pectoris.

Morbid anatomy and pathology.—Of this little is known. In fifty-three cases two had fibrosis of the left ventricle, one had fatty degeneration of the cardiac muscle, and in three there was dilatation. The frequent action of the heart in paroxysmal tachycardia may be due either to paralysis of the vagus centre, to stimulation of the accelerating centres, or to both. It is almost certain, indeed, that in most cases both of these actions happen, inasmuch as the pulse rate greatly exceeds that which occurs after complete paralysis of the vagus. The nature of the change in these nerve centres is unknown. It has been supposed by Talamon to be of a nature allied to what occurs in the cortical centres in epilepsy, and he has suggested that the fit of tachycardia may be regarded as analogous to a fit of ordinary epilepsy, the excessive action of the heart occurring in place of excessive action of the voluntary muscles. He supports this idea by the fact that ordinary epilepsy is sometimes preceded by a cardiac aura, in which palpitation, frequent action of the heart, discomfort, or even pain in the cardiac region, is felt before the epileptic fit comes on. This supposition appears to link together readily a number of facts which have been observed in regard to tachycardia. Thus the tachycardia observed in a patient suffering from tapeworm, and cured by the removal of the parasite, is exactly analogous to cases of epilepsy which have been observed from a similar cause. If this idea be correct, it might be advisable to divide the cases of tachycardia associated with organic disease of the heart from those in which no such lesion occurs, and to class the latter under the head proposed by Bouveric of essential paroxysmal tachycardia.

Symptoms.—During the attack the pulse is frequent, and may even reach the rate of 250 per minute, the face is usually pale and anxious, there is strong pulsation of the carotids, and the patient as a rule complains of discomfort about the cardiac region, sometimes with tingling down the arm. There may be no pain, and little or no affection of the respiration, but in some cases orthopnœa and a feeling of constriction or pain in the heart, which would ally the case to angina pectoris. If the attack is only a short one, no further changes are observed.

Prognosis.—The prognosis must always be guarded in cases of excessive rapidity of the heart, and in febrile diseases, if the pulse rises over 120, the risk to the patient's life rapidly increases with every rise in the pulse rate. Tachycardia due to diphtheria or alcoholism may disappear completely after some weeks or months, in the same way as paralysis of the motor nerves may disappear from other parts of the body in similar conditions.

In paroxysmal tachycardia, if reflex cause of irritation exists and can be removed, the patient may recover; but it is evident that cardiac failure might possibly set in at any time, and that any intercurrent disease is much more likely to prove fatal than when the heart is beating at a normal rate.

Treatment.—It is important to remove any cause that may lead to irritation of the heart or weakening of the vagus. Thus any undue exercise and overstrain must be prevented; quiet, both of body and mind, must be insisted upon. All mental worries and excitement should be put aside as far as possible, and the patient's nutrition maintained. In many cases the best plan of securing these requirements is the rest cure advocated by Weir Mitchell, which consists in keeping the patient isolated and in bed, so that perfect rest, bodily and mental, is maintained. At the same time abundant feeding is employed, and the appetite, which would naturally fall off from confinement in bed, is kept up by massage, which supplies the place of active exercise in this respect. In cases where the pulsations of the heart are increased by lying on the left side, the patient should lie either on the back or on the right side. All possible sources of irritation should be sought for and removed. Thus any irritation in the stomach should be lessened by bismuth or other soothing remedies; the action of the liver maintained by mercurials and salines; and regular intestinal evacuation secured by laxatives, vegetable or saline, as the case may seem to require. Cardiac stimulants, such as coffee, tea, tobacco, and alcohol, should be forbidden, and the food should be chiefly vegetable; meat being given only sparingly, if at all. During the intervals between the attacks, the nerve centres may be soothed by bromides; arsenic and iron may be given as tonics; and if the heart be feeble, quinine, digitalis, or strophanthus may be employed to strengthen as well as to steady the heart. During the attack the patient should be perfectly quiet in bed, and no exertion, either physical or mental, allowed. He should lie on his right side, as already mentioned. If any flatulence be present in the stomach, it is advisable to remove it by giving carminatives, such as spirit of ether with compound spirits of ammonia, spirit of chloroform, and peppermint water. If the tension is high, as in the case of tachycardia associated with angina pectoris, which I have already mentioned, amyl nitrite or nitroglycerin should be employed. Cold to the cardiac region, either by an ice-bag or by a spray of ether or chloride of ethyl, is sometimes beneficial, and digitalis has been recommended by Huchard at the beginning of the attack. In some cases a subcutaneous injection of morphine, one-sixth to a quarter of a grain, may give relief. Compression of the right carotid has been said to be useful, and deep forced inspiration is reputed to give relief. In cases where the attack has been prolonged, the secondary dilatation of the heart, with its attendant symptoms, must be treated if necessary by the administration of oxygen, by leeches to the præcordia, or even by venesection, in much the same way as if it were a case of mitral regurgitation.

BRADYCARDIA.

As the pulse rate varies much in different individuals, it may be difficult to fix an exact limit at which the normal pulse passes into bradycardia, but practically when the pulse rate falls below 60 a condition of bradycardia may be said to exist. Such a condition may be normal, as in the first Napoleon, whose ordinary pulse is said to have been 40 per minute. I have seen an apparently healthy man in whom it was regularly 42, but he had lived in a malarial district in the tropics many years, and this may have had something to do with the rate of the pulse. The pulse in this condition depends upon infrequency of the heart's action, and must be distinguished from the pulse which may result from such inequality of the heart's beats that some of them are too small to produce a pulsation at the wrist, and the pulse may therefore seem to be only 40 or less when the heart is pulsating at the rate of 80 per minute, or more. This is readily done by comparing the pulse of the wrist with the cardiac pulsations as heard through a stethoscope. An infrequent pulse, 35 to 60, is sometimes noticed in the puerperal condition, and the pulse is normally somewhat reduced in rate in the reaction after fever, when the temperature falls to the normal or below it.

Etiology and pathology.—Bradycardia is probably due in almost all cases to excessive action of the inhibitory nervous apparatus of the heart. This may be due to stimulation either of the nuclei of the vagus in the medulla, of its trunk, or of its terminations in the heart. It is to be borne in mind that such conditions as weaken the muscular fibre generally tend to increase the inhibitory power of the vagus upon the heart; so that stimuli which would have but a slight inhibitory action on a healthy heart, may produce an excessive effect upon one that is weakened. The inhibitory centre in the medulla may be excited directly by poisons carried to it by the blood, or by nervous lesions. Amongst poisons may be mentioned digitalis and its congeners, tobacco, aconite, biliary acids in jaundice, and possibly toxines formed in the body, as in nephritis, uræmia, and perhaps also typhoid fever, diphtheria, influenza, and pneumonia. Amongst nervous lesions may be mentioned injury to the medulla oblongata itself or to the cervical cord, to the brain by tumour or apoplexy; and a low rate also occurs in general paralysis, melancholia, and mania.

Bradycardia may be produced experimentally by strong irritation of most sensory nerves, and probably it is through irritation of the gastric branches of the vagus that the bradycardia sometimes observed in dyspepsia and gastric ulcer or cancer occurs. It is impossible to say, however, that in these cases the causation may not be a complex one, and that toxines may not play a considerable part in the production of the bradycardia, as well as the reflex irritation from the abdominal organs.

Irritation of the vagus trunk will also produce bradycardia. This was observed by Czermak, who was able to press his vagus between his finger and a hard lump in his neck, which in his paper he called a hardened cervical gland, but which the late Professor Sharpey told me was really an exostosis on one of the cervical vertebræ. On doing this he not only noticed that his heart became slow or stopped entirely, but that he got a feeling of intense constriction or compression on the chest, to which he applied the term "Beklemmung," which signifies that the feeling was such as would be produced by a giant's hand compressing the thorax. Bradycardia may result from irritation of the vagus trunk by tumours, and possibly also from neuritis. Neuritis of the vagus may be the cause of the low pulse rate sometimes observed after diphtheria, influenza, typhoid fever, and in pneumonia and diabetes. As I have already mentioned, weakness of the heart tends to increase the action of the vagus upon it, and bradycardia occurs in cases where the muscular fibre of the heart has undergone degeneration either of a fatty or fibroid nature. It is observed also in aortic obstruction and emphysema. In emphysema it is possible that the slowness is partly due to reflex irritation from the lungs, but probably it is largely dependent upon the increased pressure which the right heart has to overcome. The infrequency which occurs normally after fevers, more especially influenza, diphtheria, typhoid fever, and pneumonia, is probably due in part to reaction after the increased pulsations which the heart has had to maintain for several days or weeks under the influence of a raised temperature; partly to the weakness consequent upon this increased action as well as the action of the high temperature itself upon the cardiac muscle; and also possibly to the effect of toxines circulating in the blood. Where there is increased resistance to the action of the left ventricle, bradycardia may also be observed, as in cases of lead poisoning and in aortic obstruction, as already mentioned.

Prognosis.—A pulse that is somewhat below the average rate, but is steady and regular, is sometimes coincident with an unusually long life; but in cases where the pulse is much below the normal, the prognosis is usually grave, especially if there be any indication of cardiac weakness, or if the patient be advanced in years.

Treatment.—The indications for treatment are to increase the nutrition of the heart, and remove if possible any irritation affecting the vagus nuclei, trunks, or endings in the heart, and to increase as far as possible the nutrition of the cardiac muscle. For the purpose of increasing such nutrition, the food should be abundant and digestible, and the elimination of waste products should be increased as much as possible by the use of purgatives, diuretics, or sudorifics. Where poisons or toxines are present, an endeavour may be made to antagonise their action; thus digitalis and drugs of a similar nature may be to a certain extent counteracted by strychnine. Where the bradycardia is due to reflex action, this may sometimes be diminished by large doses of bromide of potassium; and when other means fail, belladonna or atropine in sufficiently large doses will paralyse the inhibitory action of the vagus upon the heart. In cases where the bradycardia has come on suddenly, apparently from some reflex irritation, this treatment may be useful; but where it is of a persistent nature, the full action of belladonna cannot be kept up. Where there is evidence of increased vascular tension, nitroglycerin or other nitrites may be employed, and the diffusible stimulants, such as alcohol, ether, and sal volatile, may be administered for the relief of faintness, or where there is any increase in the habitual slowness of the pulse. As thyroid extract tends to lower blood pressure and quicken the pulse, its use would appear to be indicated in bradycardia. In the only case in which I have tried it, however, the patient died before there was time to notice any effect of the treatment

DISTURBANCES OF RHYTHM.

The cardiac rhythm may be disturbed either by the apparent omission of a beat, or by the intervals between successive beats being of different instead of the same length. To the former condition the name of intermittence is applied, to the latter that of irregularity. As I have already mentioned, the unequal beating of the heart in either of these conditions tends to induce inequality in the strength and volume of the pulse as well as in its rhythm.

Pathology.—Irregular cardiac rhythm may, like bradycardia, be due to irritation of the vagus roots or ends in the heart. It is rarely, if ever, associated with lesion of the vagus trunk. Its causation is well shown in cases of poisoning by digitalis, as illustrated by some tracings which I took in 1865. The first tracing was taken shortly after the patient had begun to recover from a condition of collapse in which the pulse was very rapid. It shows rapid pulse with a few slow beats, and the next one a slow pulse with one or two quick beats, the third a regular slow pulse, and the fourth a fairly regular pulse of moderate rapidity. Irregularity may also be due to a central nervous lesion, to poisons circulating in the medulla, to reflex disturbance, or to changes in the heart itself. Amongst the central causes are increased intracranial pressure from tumour, abscesses, hæmorrhage, or inflammation, tuberculous or otherwise. It occurs also in epilepsy, concussion, and various kinds of mental derangement. Amongst the most common toxic agents which produces it are tobacco, tea, and coffee, used as stimulants, and aconite, belladonna, and digitalis as medicines, and unclassified products of tissue waste in chronic gouty conditions.

Reflex irregularity may occur from irritation from the stomach, liver, lungs, and kidney, one of the most common causes being distension of the stomach by food or flatus, or irritation of it by unsuitable articles of food.

Amongst the most important changes in the heart itself which lead to intermission are dilatation and degeneration, fatty or fibrous. With these may be associated sclerosis of the coronary arteries, either as they leave the aorta or as they pass along the ventricular walls.

Symptoms.—Intermittence of the pulse may occur either at regular or irregular intervals. Sometimes the intermittences are far apart, at other times they are near together. Thus a pulse may intermit once in twenty or thirty beats or even more, at others it may intermit every third or fourth beat with a fair amount of regularity, while sometimes intermission will occur once in two seconds, next in ten, next in three, next in five, and so on, without any regular order. Sometimes two successive beats will come close together, and be succeeded by a long interval. To this kind of pulse the name of bigeminal has been given; when three beats come together followed by an interval, it is called trigeminal. The rhythmical occurrence of two or three beats in such case is probably connected with the respiration. In the so-called pulsus paradoxus, the pulse during inspiration becomes quicker and smaller, while in expiration it becomes slower and larger. This condition is merely an exaggeration of what occurs in health during very deep inspiration and expiration, but the name pulsus paradoxus is applied to it only when it becomes so much exaggerated as to be readily perceptible to the finger, and to occur on ordinary respiration instead of forced respiratory effort. Occasionally, in extreme cases, the pulse may be so small as to become imperceptible. Irregularity in force and rhythm may be so great that it is impossible to count the pulse in a case of so-called delirium cordis.

Prognosis.—Special significance has been attached to the bigeminal pulse. It has been looked upon as of grave import; but I have two tracings showing this pulse in a healthy man, who is at the present time still alive and well. I have known also a case of great irregularity of the heart last for fifty-seven years in a lady who died suddenly of syncope at the age of 84. Irregularity of the heart, if associated with evidences of cardiac weakness, and especially when the patient is beyond middle life, is of grave import.

Treatment.—In cases where the irregularity is dependent upon cerebral mischief, the treatment must be adapted to the condition, and such surgical or medical means may be used as are likely to remove it, if possible.

When the irregularity is due to an overloaded stomach, this may be relieved by an emetic of mustard and water, or by the gastric syphon with the subsequent administration of bismuth, hydrocyanic acid, and morphine, or even better, belladonna, if necessary. If the distension be due to flatulence, it must be removed by carminatives. Where there is evidence of hepatic disorder, a mercurial pill followed by a saline is best.

In cases of irregularity from toxic agents, lessened indulgence in tobacco must be insisted upon, and sometimes the abstinence must be complete. It has seemed to me that where cardiac irregularity has been brought on by the use of tobacco, a very small quantity daily will keep up the cardiac disorder, and nothing but total abstinence from the weed will cure it. In other cases tea or coffee causes the mischief, and must be abstained from completely. In cases where toxines are probably formed in the body, an eliminative treatment by mercurials and salines is to be followed. One of the most important points in the treatment of this condition is to remove mental worry or strain; and when this cannot be done, the effect of these disturbing influences on the heart may be somewhat lessened by the administration of bromides, especially if combined with a small quantity of salicylate of soda. The nutrition of the cardiac muscle must be maintained by appropriate diet and removal of waste, as already mentioned under bradycardia; and in cases where the disturbance of rhythm is only occasional, it may be relieved for the time being by diffusible stimulants, such as alcohol, especially when given in hot water. Where the attacks come often, it is highly desirable to avoid the administration of alcoholic stimulants, because the alcoholic habit may be thus acquired, and in place of them half a teaspoonful of sal volatile, either in plain water or peppermint water, may be given.

PALPITATION.

This is a feeling of frequency or force of the heart's beat, which may be only subjective, but usually is objective also.

Palpitation is in its essence a sensory affection and not a motor one, for in hypertrophy and organic disease of the heart we may have the cardiac pulsations greatly increased both in force and frequency without this attracting the patient's attention. On the other hand, in some nervous or hysterical conditions the patient may complain of palpitation when no increase either in force or in rate is perceptible to an observer.

The heart is largely influenced by the central nervous system, and more especially by that portion of it the site of which has not been located, but which is the seat of the emotions. So much is this the case, that the heart is often regarded as the seat of the emotions, and in popular language they are expressed in terms of its action. Thus it is common to say, "His heart beat high with hope," or "His heart sank within him for fear." Some of the most powerful emotions are those which are connected with the relations of the sexes, and these above all others give rise to palpitation.

Etiology.—Nervous palpitation, apart from organic disease of the heart, is more common in women than in men, and it is more apt to occur at the time when the sexual organs are either undergoing development at puberty or retrogression at the menopause. It is more liable to occur also at the time of the menstrual period. Any condition of weakness, by lowering the regulating apparatus of the heart, may induce it; and thus it is more liable to come on in persons who are convalescent from acute disease, who are debilitated by overwork, insomnia, anæmia, chlorosis, hæmorrhage from any cause, over-lactation, and more especially in those who have suffered losses of any kind from the sexual organs, or from sexual excess in either sex. It may be due also to local weakness of the heart, or to poisons circulating in the heart itself and in the nerve centres which regulate its action.

Amongst the exciting causes we find external heat as on a hot day, exposure to hot air as in a Turkish bath, or a stay, especially if prolonged, in a hot bath. It is frequently influenced by position, so that when the heart is at all irritable the patient may be unable to lie on the left side. The reason of this is, that the heart, being a more or less mobile organ, tends to fall to the left when the patient is lying on that side, and the blow which it receives from the ribs as it strikes against them at each systole acts as a stimulus to increased contraction. In this condition an external stimulus is the cause of the palpitation; but the stimulus of undue pressure or over-exertion to the inside of the heart may lead to palpitation, and once induced, this may be kept up by the normal arterial tension or pressure. This condition, to which the name of "irritable heart" has been given, is observed frequently in young soldiers, probably from the strain of carrying the knapsack and other accoutrements; and I have seen it also in men of middle age who are accustomed to do very heavy work, such as coal-heavers and stevedores.

One of the most common causes of nervous palpitation is a condition of chronic poisoning by tea, coffee, tobacco, or alcohol; and possibly various toxines formed in the body may be partly to blame for the production of palpitation in the debilitated conditions already mentioned, as well as in chronic dyspepsia.

Reflex irritation is a powerful exciting cause of palpitation, and the stimulus which produces it may start from any one of the abdominal organs, such as the stomach over-distended by too full a meal or by flatus, or irritated by the products of the decomposition of food or the acids formed from sugar or alcohol. Irritation in the intestines by fæcal masses or by parasites, and calculi in the gall duct or kidney, also lead to palpitation. A floating kidney sometimes gives rise to palpitation of a very marked character indeed, and it may be associated with uterine and ovarian troubles of many kinds; it generally occurs in exophthalmic goitre.

Symptoms.—The symptoms are those which one connects with irritation of the vagus combined with dilatation of the peripheral arteries. Thus there is a feeling of oppression or anxiety, sometimes even of pain, in the chest, which I have already mentioned as consequent upon compression of the vagus, while there is a violent pulsation of the heart, fluttering in the chest, throbbing in the arteries, sometimes increased quickness of the pulse and flushing of the skin and a feeling of heat in the surface, such as accompanies peripheral dilatation of the vessels.

Prognosis.—Nervous palpitation in itself does not tend to shorten life. If associated with organic disease, the prognosis will depend upon the lesion and not upon the palpitation. The duration of the palpitation, however, will depend very greatly upon the conditions to which the patient is subjected. If the causes which give rise to it are removed, recovery may be rapid, but otherwise the patient may be liable to palpitation for several years.

Treatment.—Instinct has taught people who suffer from palpitation that it can almost always be relieved by pressure on the cardiac region, and so they naturally put their hand over the heart to still its beatings. Pressure may be applied in much the same way, but more constantly, by a plaster over the cardiac region; and a belladonna plaster has been found by experience to be on the whole more advantageous than plasters of other kinds. How far the good effect of this is due to the pressure and how far to the warmth afforded by the hand or the plaster, it is somewhat difficult to say. Internally, a small quantity of neat brandy will frequently relieve the palpitation very quickly; but this is a dangerous remedy, and should never be prescribed if it can be possibly avoided, while patients ought to be warned against it, as it is very likely to lead to the alcoholic habit. The frequent repetition of small doses of alcohol lessens the regulating power of the nervous system over the heart, and thus the continuance of the remedy makes larger and larger quantities of it necessary. Sal volatile is as good as brandy, and has this great advantage, that an overdose will probably make the patient sick, and thus the habit of nipping is not likely to be acquired to anything like the same extent. Sometimes a glass of cold water slowly sipped, or of effervescent water, will have a somewhat similar effect. Failing this, some thin hot soup or beef essence mixed with hot water will lessen the palpitation. In cases where the patient is anæmic and debilitated, iron, nux vomica, and hypophosphites or glycero-phosphates are useful. Bromides may be employed to quiet the irritability of the nervous system. To remove any irritating substances from the intestine or liver, aperients and cholagogues, such as a blue pill and subsequent saline, should be used, and any irritability of the stomach or intestine may be corrected by alkalies, bismuth, and pepsin if necessary.

In the case of an irritable heart I have found belladonna of great service when given in pretty large doses so as just to produce slight dryness of the mouth. But in one patient, a stevedore, where I had apparently cured the irritability, and the man returned to work, the heavy exertion brought the malady on a second time, and then belladonna seemed to be powerless against it. In such cases, therefore, absence from bodily strain should be insisted upon. As emotions are most powerful to produce palpitation, these should be quieted as far as possible by withdrawing the attention of the patient from the circumstances which cause the emotion; but where this cannot be done, small doses of salicylate of soda, say 10 grains, with 10 to 20 of bromide of potassium, two or three times a day, or double this dose at bedtime, are useful.

In the case of floating kidney, the organ should either be supported by a pad and belt or by a truss, or else should be fastened to the abdominal wall by surgical operation. In cases of palpitation and flushing occurring at the menopause, the administration of tablets of ovarian tissue have sometimes proved useful. Last but not least, small doses of digitalis, 5 minims of the tincture, three times a day, or just when the attack comes on, tend to quiet and to lessen the palpitation.

ANGINA PECTORIS.

A paroxysmal attack of pain or intense oppression in the cardiac region, usually accompanied even in its slighter forms by necessity for rest, and in its more severe forms by a feeling of impending death.

Typical angina pectoris is characterised by certain well-marked symptoms, just as is a typical attack of gout; but in the same way as gout may vary from intense and hardly endurable pain to a mere twinge in the joint scarcely deserving of attention, so angina pectoris may vary from the most intense pain and anguish down to a slight discomfort in the chest, hardly deserving of the name of pain. It is to be remembered, also, that all pains in a joint are not gout, and all pains in the heart are not necessarily of the nature of angina. Moreover, it is not to be forgotten that the seat of the perception of pain is the brain; and although usually such a perception is excited by peripheral irritation, yet the sensation of pain may occur without any peripheral change, and hysterical patients have sometimes uttered such loud complaints of pains in their knee-joint that the leg has been amputated without any mischief in the joint being found. In like manner young hysterical patients may have pains in the heart without any organic change, and without there being any risk whatever to life.

Etiology.—Functional angina occurs in young people of a nervous temperament, and especially in hysterical families. Organic angina is more common in men above 45 years of age, although it has sometimes been observed in younger people. Amongst the marked predisposing causes may be mentioned gout, syphilis, and rheumatism. The first leads to high tension, the second causes atheroma, and the third induces organic change in the heart and vessels. Amongst the most powerful of predisposing causes should be reckoned worry, anxiety, and grief, as well as sudden severe exertion in persons unaccustomed to it.

Exciting causes.—The exciting causes of angina are such things as will raise the blood pressure, more especially excitement or exertion. All kinds of excitement are not equally injurious; pleasure, hope, compassion, love, or even joy, do not tend, except when very excessive, to bring on pain, while anxiety, sorrow, fear, and more especially anger, tend to bring on an attack. Such exertions as increase the blood pressure above the usual, as going uphill, going upstairs, lifting a weight, strapping a portmanteau, or running quickly to catch an omnibus or train, are exceedingly apt to bring on an attack. Sudden stimulation of a sensory nerve by painful impressions has the effect, as is well known, of raising the blood pressure, and it also will tend to cause an attack. The same consequence occurs from the sudden application of cold, either to the surface or to the stomach, as these also tend to raise the blood pressure; and distension of the stomach itself by food or by flatus may have a similar effect.

Pathology.—Many views have been held regarding the pathology of angina pectoris. Some, like Romberg, have regarded it as a neuralgia of the heart; others, like Trousseau, as a form of epilepsy; and much discussion has occurred as to whether death in fatal cases is due to spasm or distension. In all probability the pain of angina pectoris is caused in the same way as pain in other hollow organs consisting of involuntary muscular fibre. In all these, extreme pain is produced by combination of distension from within, and of spasm of the muscle to which it leads. Thus pain in the bladder is caused by distension, along with contraction of the vesical walls, which are quite unable to evacuate the urine on account of the obstacle opposed by stricture or spasm of the neck of the bladder. In colic the pain is caused by spasmodic contraction of the muscular fibres, generally accompanied by distension by flatus. In renal and biliary colic the pain is produced by the passage of a calculus too large for the lumen of the duct, which thus causes both distension and spasm. Perhaps the most exact analogy, however, is to be found in the pain of migraine, where, as I have found and described in my own case, there is peripheral contraction of the vessels with proximal dilatation: so that, while the carotids are distended and throbbing violently, the temporal artery may be so constricted as to feel like a piece of piano-wire under the finger, and the pulse in it would be barely perceptible. In migraine the relief of tension by compression of the carotid at once relieves the pain in an aching artery, and in the case of angina pectoris relief of tension by dilatation of the peripheral vessels relieves the pain of the aching heart.

In the healthy body, as I have already mentioned, there are certain nervous mechanisms by which the pressure in the heart regulates the size of the vessels, and the pressure within the vessels regulates the beat of the heart. In angina pectoris this relation appears to be completely disturbed: and in a case which I described more than thirty years ago, the tension in the vessels was greatly increased, but instead of the heart becoming slower, as it would in the normal condition, it beat much more quickly than usual, just as if its inhibitory apparatus had been completely paralysed, and was simply responding, like an excised heart, with quickened beats to the increased pressure within it. But just as we find that, in cases of headache, disordered innervation of the vessels usually depends upon some local disturbing cause, such as a bad tooth, a strained eye, an obstructed nostril, or an inflamed ear, so in cases of angina pectoris we find that the nervous disturbance is usually accompanied by a local change, and this change is of a nature which becomes of the utmost importance, because it affects such a vital organ as the heart.

The essential lesion in angina pectoris is weakening of the heart, and more especially such irregular weakening as may be produced by atheroma of the coronary arteries. Where the heart is generally weakened, either by the continuance of some febrile disease, such as typhoid fever, phthisis, or malaria, or where general fatty degeneration has occurred, the patient may remain free from any attack of angina pectoris; but where atheroma of the coronary arteries has taken place, angina pectoris is very likely to come on. The first explanation, although it may possibly not be the true one, of such an occurrence is that the different parts of the heart have not all been weakened to the same extent, and thus irregularity of the different parts of the ventricles or auricles may tend to cause pain, which would not have occurred if they had all been acting together in unison, however weak their action might have been.

Little or nothing is known regarding the afferent nerves of the heart, through which painful sensations are conveyed from it to the sensorium, although it is probable that some of the fibres of the vagus convey these sensations, inasmuch as when one of the vagus trunks is divided, irritation of its central end will produce a reflex action upon the heart through the other vagus, and also upon the vessels through the vasomotor centre. The feeling of compression or oppression upon the chest is hard to describe, but it can be easily understood by any one who cares to try the experiment by pressing deeply over the carotid artery on the right side. In my own case this feeling of oppression becomes so great that after about a minute I am obliged to cease the compression. A similar feeling of constriction or oppression may be caused by painful emotions, and will continue to be felt as a physical sensation after the emotion which caused it has passed away. This fact I can vouch for also from my own experience.

We find, then, that in angina pectoris we have the symptoms of irritation of the sensory fibres of the vagus combined in some cases with evidence of temporary paralysis of the inhibitory fibres, and at other times, in cases where the pulse is exceedingly slow, apparently with over stimulation of the inhibitory fibres. In some cases, as in the celebrated one of John Hunter, who died of an attack of angina pectoris caused by anger, emotion proceeding from the nerve centres may lead to alterations in the action of the heart. In still more numerous cases, however, the attack is brought on by sudden exertion, the first effect of which is to raise the pressure within the whole aortic system, and consequently also in the left ventricle. When the exertion is taken immediately after a meal, at which period, as I have already stated, the blood pressure is usually above normal, the attack of angina pectoris is still more easily brought on. Distension of the stomach by flatulence will also bring on the attack, which frequently passes off as soon as the flatulence is relieved. In this case it is impossible at present to say whether the attack is due to reflex irritation of the heart or vessels through the nerves of the stomach, or whether actual physical displacement of the heart by the distended stomach is the true cause of the disturbance of circulation, and consequently of the pain. In most cases an attack of angina pectoris is brought on by such causes as lead to excessive pressure in the left ventricle. I believe, however, it is possible that excessive tension in the right ventricle may have a similar action, but this will be discussed more fully under the head of "Cardiac Asthma."

Atheroma of the coronary arteries, more especially of the left coronary artery, may, by obstructing its lumen, cause angina pectoris without any other observable alteration in the aorta or in the valves of the heart; but where atheroma is present, it is very likely to be associated with disease of the aortic valve, or with dilatation or aneurysm of the aorta itself, as well as with fatty degeneration of the cardiac muscle, due to imperfect blood supply. It is evident that a heart thus weakened will be all the more likely to suffer if the pressure against which it has to work is increased above the normal; and as this condition occurs as a consequence of gouty kidney, angina pectoris is very apt to occur in gouty people.

Symptoms.—In a typical attack of angina pectoris the patient is suddenly seized by a severe pain in some part of the cardiac region; sometimes near the lower end of the sternum or at the apex, but occasionally at the base of the heart. The pain sometimes appears to strike through to the back, or up the neck and down one or other arm, but especially the left, generally on its inside. The pain is accompanied by a peculiar sensation of oppression, which is described in different terms by different patients. Some compare it to pressure from without, as if the chest were constricted by an iron band or by a heavy weight pressing upon it, while others speak of it as distension, as if the heart were distended and going to burst, and in severe cases the patient feels as if death were impending. The patient is unable to move on account of the pain, and feels difficulty in taking a deep breath. Hyperæsthesia of the skin over the painful areas is often found. This has been carefully studied by Mackenzie and Head. The face and skin generally are pale, cold to the touch, and often covered with a clammy sweat. There is no real obstruction to respiration, and the lungs are quite clear, with free entry of air during the attack. In some cases the pulse is said to be quite unaffected, in others it is said to be slower than usual, and in others again it is very quick. The attack lasts for a time varying from a few minutes to several hours, and may end either suddenly or, more generally, gradually, the paleness and coldness passing off and being succeeded either by the normal condition or by flushing and warmth. In slighter cases, especially at the commencement of the disease, there may be nothing but a little discomfort or pain felt over the sternum on a sudden movement, and especially on walking up a hill or upstairs. At first this pain disappears as soon as the patient stops, but at a later stage it may continue for a minute or two after the exertion has ceased, and then pass away. When the pain is not very severe, the patient may be able to continue walking, and if he does so, the pain will generally go away and remain away while the exercise is continued; but it will probably return again when the patient commences to walk up hill after a prolonged rest. Frequently the expulsion of some flatus from the stomach is accompanied by marked relief or complete disappearance of the pain. Any mental excitement, more especially anxiety or anger, may bring on an attack of pain in the same way as bodily exertion. In nervous or hysterical persons, and especially in young women, pain in the heart may come on suddenly, and may even be accompanied by a sense of oppression in the chest; but in them it does not usually radiate in the same way, and it is frequently accompanied by a choking sensation like that of the globus hystericus, the feeling of suffocation being felt higher up than the constricted feeling in true angina. The name of pseudo-angina has been given to such cases.

Varieties.—The pain and even the feeling of constriction in angina are not diseases, but only symptoms, just as cough is a symptom of irritation in the respiratory passages. Cough may indicate very different pathological conditions. It may occur in a trifling catarrh, from which the patient will speedily recover; in chronic bronchitis and emphysema, which will make the patient an invalid for years; or it may indicate the presence of advanced tuberculosis, from which the patient will speedily die. In like manner, pain in the heart may indicate a neurosal affection which is not at all dangerous to life; or it may indicate a serious disease of the most dangerous character.

Angina pectoris is therefore to be divided into two classes, *functional* and *organic*, the former not being serious and the latter being very dangerous. Just as it may not be always easy to say whether a cough is caused by simple catarrh or by the presence of tubercle, so it is not always easy to distinguish between the pain due to true and false angina, but the former occurs most frequently in subjects below 40; it is commoner in women than in men; the pain is less severe, is not accompanied by a sense of impending death, and there is a feeling of dilatation rather than of compression in the thorax. These points of distinction are not always trustworthy. For example, the pain in the angina of a hysterical girl may be much more severe than in an early case of true angina in an elderly patient with commencing atheroma of his vessels. Hysterical or neurosal angina may occur in patients with valvular disease, but the presence of atheroma, fatty degeneration of the heart, or evidences of failing compensation when valvular disease is present, strongly indicate that pain in the heart evidences the presence of true rather than of pseudo-angina.

A useful subdivision is, according to the causation of angina, into —*neurosal; reflex; toxic.*

It is difficult to say whether the pain in the hysterical or neurosal variety is caused by spasm of the vessels or not; but in both the reflex and the toxic varieties it is probable that the blood pressure is raised, although we are obliged to reason a good deal from analogy, and have but very few exact observations on the condition of the vascular system during attacks of angina.

The reflexes which tend to cause anginal attacks are, as I have already mentioned, cold to the skin or to the stomach, painful impressions upon sensory nerves, distension of the stomach by food or flatus; and to these may be added irritation of the respiratory passages by the sudden inhalation of cold air, as when going out of a warm room on a cold and frosty morning.

The most important cause of toxic angina is tobacco, although excessive use of tea or coffee may also produce it. The active principles of these substances, namely, nicotine and caffeine, have been found experimentally to cause contraction of the peripheral vessels and rise of blood pressure. In addition to this, nicotine has got a special action upon the vagus, stimulating its roots, and in very large doses paralysing its peripheral terminations in the heart. In consequence of this, it tends to produce in moderate doses slowness of the pulse, but in excessive doses, either irregularity of the pulse or excessive rapidity. Disturbance of the heart's rhythm is much more common than angina from tobacco, and generally precedes or accompanies the attacks of angina. Other symptoms of nicotine poisoning usually occur also, such as tobacco amblyopia, dyspepsia, headache, giddiness, and confusion of thought. The attack is often associated with those symptoms of tobacco poisoning which are very clearly seen in those who have smoked a cigar for the first time, namely, a feeling of oppression at the chest, giddiness with extreme faintness: the skin is very pale, cold, and often covered with clammy sweat. All the symptoms just mentioned usually disappear quickly when the use of tobacco is discontinued.

Diagnosis.—The diagnosis of angina depends, first of all, on its seat in the cardiac region; secondly, the character of the pain, which in severe cases is accompanied by a feeling either of great oppression or distension; thirdly, on the radiation of the pain through to the back, up to the neck, and down the arms, especially the left arm; fourthly, on its spasmodic character; fifthly, on the concomitant symptoms, namely, the coldness of the surface, pallor of the skin, sometimes with cold sweats and occasionally with some lividity, and inability to move for fear of increasing the pain. The seat of the pain helps to distinguish between angina and pain in the stomach from over-distension, although the pain in both conditions is greatly relieved by the expulsion of flatus. The situation helps also to distinguish angina from biliary or renal colic, as the pain caused by calculus in the left kidney shoots down towards the genitals instead of upward or through to the back. Biliary calculus may cause pain, coming on quite as suddenly, leaving off as suddenly, and being quite as acute while it lasts as angina pectoris; but while angina tends to make patients remain absolutely still, the pain of biliary calculus renders them unable to keep quiet. Radiation of the pain in some cases of biliary calculus is not unlike that of angina, as it may shoot through to the back, round on either side, and upwards into the neck, but it tends to radiate more to the right shoulder than down the left arm. At the same time, I think that biliary colic, due to small calculi or to the passage of inspissated bile, is sometimes mistaken for angina pectoris.. More especially is this the case when the calculi are just sufficiently large enough to distend the cystic duct and cause intense pain on their way through it, but are not sufficiently large to obstruct the common bile duct, and thus they do not lead to jaundice. In such cases a careful examination of the liver will probably show considerable tenderness over the gall bladder, while the heart itself may be perfectly healthy. Should, however, the valves of the heart be diseased, in such a case it would be very difficult indeed to come to a satisfactory diagnosis, although the tendency of the biliary calculi to produce movement instead of quiet, and the absence of a sense of impending death, might enable a correct conclusion to be arrived at. Cases of intercostal neuralgia sometimes lead to a fear of cardiac disease on the part of the patient, but in such cases the pain is not of a spasmodic character, and generally tender spots are to be found on pressure along the dorsal vertebræ and in the course of intercostal nerves.

Prognosis.—The prognosis in hysterical and neurosal angina is good both in regard to their complete cure and to the absence of risk to life. In true angina, on the contrary, the prognosis is grave, for however slight the attacks may be at first, they are likely to occur with ever-increasing intensity, and finally to prove fatal. In some cases the first attack may be likewise the last, and the patient may die suddenly, without having had any previous symptom. Much more frequently, however, the disease comes on gradually. Slight pain in the chest on walking uphill or on excitement is noticed by the patient, but this is often so slight that he pays little attention to it, and may not mention it either to his friends or to his medical adviser. As the disease advances, the pain is brought on by slighter causes, and after a time will come on without any apparent cause.

Treatment.—The indications for treatment are, during the attack, to lessen the resistance the heart has to overcome, as well as to strengthen its contractions, and, during the interval, to strengthen the heart and improve the nervous mechanism connected with it, and to prevent the patient from being exposed to those circumstances which are likely to bring on an attack. The patient should be enjoined to avoid all unpleasant excitement or all undue exercise, but gentle, steady daily exercise, not severe enough to bring on an attack, is of advantage. He should always rest for some time, say half an hour or more, after meals. The patient should not be forbidden to walk, but he should be strictly advised not to walk so quickly along the level nor to walk uphill in such a way as to bring on an attack. Exercise short of this may be permitted with advantage. He should live as quietly as possible, avoid late hours, and especially any unpleasant excitement. He should have regulated exercises to bring the various muscles of the body into action. Exercises well adapted for this purpose are those which are used at Nauheim in the treatment of heart disease. The object here is gradually to increase the strength of the heart by giving it a little more to do every day, but great care must be taken that no excess occurs on any one day, as not only might this prove dangerous to the patient at the time, but would in any case throw him back, and he would lose the advantage already gained.

As the process of atheroma is the most potent cause of all in the production of angina pectoris, it requires to be especially treated. In its nature it is like the inflammation of joints, so that Virchow has given to it the name of "arteritis deformans," thus linking it with arthritis deformans. In both these forms of inflammation, iodide of sodium or potassium is a useful remedy, and in commencing angina pectoris it ought certainly to be employed if the patient will bear it. The powers of different patients to stand this drug vary extremely, and where some patients will take 10 to 20 or even 30 grs. three times a day without a word of complaint, 2 grs. will produce in others such depression, discomfort, and misery, that one is obliged to give up the use of the drug. It may be given along with aromatic spirit of ammonia to counteract the depressant tendency of the iodide. For example, 5 grs. of iodide may be given with 15 to 20 minims of aromatic spirit of ammonia three times a day, and if the patient will stand it, this may be doubled or even trebled. It is sometimes a good plan to give the iodide for a fortnight or three weeks at a time, and then to leave it off for a similar period, when it is again resumed and again left off. This treatment should be continued for months or even years. In cases where a small dose is not well borne, the patient will sometimes cease to suffer from coryza and discomfort by the dose being increased; so that where 2 or 5 grs. may make the patient miserable, the same person may be able to take 15 or 20 grs. twice a day without any discomfort.

The most powerful drugs that we have at our disposal for lowering the blood pressure are the nitrites, either inorganic or organic. Nitrates also have a similar action, but it is slower and more persistent. In order to keep the blood pressure low, nitroglycerin, which is a nitrate although it behaves more like a nitrite, may be given in the form of tablets or of a 1 per cent. solution three times a day or oftener. The usual dose is $\frac{1}{100}$ gr., which is contained in one tablet or one minim of the pharmacopœial solution. Nitrite of soda has a similar action, but it has not found much favour in practice. It is used in doses of 1 or 2 grs., and is prepared also in the form of tablets containing 2½ grs. each. One of the most useful remedies for reducing the blood pressure is nitro-erythrol or nitro-erythrite. This may be given in the dose of half a grain in the form of a tablet three times a day, and gradually increased until 3 or 4 grs. or even a larger quantity are taken for a dose. Manitol nitrate in doses of 1 gr. has been also employed, but has not come into general use. Nitrate of potash in 10- to 20-gr. doses three times a day tends also to lower the blood pressure, but is less active than the substances just mentioned. A diet sparing in quantity and as nearly as possible vegetarian in quality tends to lower the pressure, and thus to afford relief to the patient.

During the attack the pain is usually cut short most quickly by the inhalation of nitrite of amyl or nitrite of isobutyl. These drugs sometimes give relief much more certainly when they are freshly prepared than if they are kept long. They may either be inhaled from a bottle, which can be carried about with the patient in the waistcoat pocket, or they may be enclosed in glass capsules covered with silk, which are simply crushed and the vapour inhaled whenever the paroxysm comes on. I have never seen any bad results from the inhalation of nitrite of amyl, although on one occasion I pushed it until the patient began to become slightly livid. This I did in 1867, shortly after I first recommended the drug for use in angina pectoris. The case in which I used it was one of pseudo-angina in a hysterical young woman, and the inhalation gave no relief whatever, so that I was led to push it very far in the hope of obtaining a good effect. Nitroglycerin has a similar action, but is not so rapid. It is, however, more convenient, as it can be carried about in the form of tablets in a small box in the patient's waistcoat pocket. The best way to use them is to let the patient slowly nibble one or more tablets until the pain disappears. Chloroform also gives relief, but usually it does not do so with the same certainty and rapidity as nitrite of amyl. The plan recommended by Balfour is to drop the chloroform on a sponge in a smelling-bottle, from which the patient inhales. If no more chloroform be put on the sponge than what it can suck up, there is no risk whatever from the employment of the anæsthetic in this way, as the moment it begins to take effect, the patient's hand drops and the inhalation ceases. Should, however, the bottle contain more chloroform than the sponge will suck up, and the patient be using it in bed, the pillow might become soaked with the chloroform which runs out, and fatal anæsthesia ensue from the patient's head falling down upon it. In cases where these remedies do not succeed, the hypodermic injection of morphine from a quarter to a third of a grain may be employed.

Hot-water bags or bottles over the cardiac region, to the nape of the neck, to the feet, and to the hands, are useful, and a carminative mixture to bring away flatulence should also be given. A useful formula consists of 15 minims of aromatic spirit of ammonia, 10 minims compound spirit of ether, 10 minims of spirit of chloroform, 10 grs. bicarbonate of soda, and 1 oz. of peppermint water. This mixture is also useful in cases of functional angina, where flatulent distension in the stomach often plays a great part in the attack.

CARDIAC ASTHMA.

Sudden and great difficulty of breathing coming on upon slight exertion, or with no apparent reason, and not depending upon interference with the entrance of air into the lung.

I am inclined to think that a good deal of the confusion that at present exists in regard to the varieties of angina pectoris depends upon the fact that cardiac asthma and angina pectoris may coexist. In angina it is usually the left side of the heart that is more especially involved, and high arterial tension is the most powerful exciting cause. As I pointed out long ago, excessive tension in the right side of the heart may possibly cause pain as well as in the left, but, as a rule, distension of the right ventricle does not so readily occur as distension of the left, for the tricuspid valves yield more easily, and allow regurgitation into the large venous trunks. Dilatation of the right ventricle is, however, closely associated with dyspnœa; and any great exertion, even in healthy people, causes the right ventricle to become distended, and the apex-beat to become imperceptible at the ordinary position, and pulsation is felt in the epigastrium. Shortness of breath on exertion is a very frequent concomitant of cardiac weakness and cardiac disease, but sudden and alarming spasms of dyspnœa are usually dependent upon weakness of the right ventricle. We do not often come across a case in which the condition of the right and left ventricles is entirely different, and I may perhaps therefore be pardoned for describing one which I think illustrates the pathology of cardiac asthma better than any general description.

N. B., æt. 66, of short but of stout build, complained much of breathlessness and sleeplessness. His urine was of a specific gravity of 1028, clear, acid, free from sugar, but with a trace of albumin. His appetite was poor, his bowels regular. The liver was full and tender, the cardiac impulse was in the fifth interspace in the nipple line, and a slight systolic murmur was audible at the apex, and another slight systolic murmur was audible at the second right costal cartilage. Both murmurs were slight, and were but very little propagated in any direction. The second sound was a little blurred, so I did not feel sure whether there was not a diastolic murmur over the sternum. The apex-beat was forcible and heaving; the lungs were clear, with the exception of slight crepitation at both bases behind. As the patient entered my consulting-room, I was struck by his exceeding shortness of breath, which the slight exertion of walking across the passage had brought on. The strong pulse, the enlarged heart, and powerful heaving impulse of the left ventricle, showed that it was not enfeebled, while the free entrance of air into the lungs showed that the dyspnœa was not dependent upon defective respiration. I therefore came to the conclusion that the right ventricle was weak, and my diagnosis, as recorded in my case-book, was that the patient was suffering from "probably interference with the right ventricle by calcification of the coronary artery." The patient died suddenly eight days afterwards. On post-mortem examination, some fluid was found in the right pleura, and also in the pericardium. The left ventricle was much thickened, the walls being apparently almost three times as thick as the normal. The right ventricle, on the contrary, was exceedingly thin and soft, so that one could push a finger through it more easily than through a sheet of blotting-paper. I had expected to find an atheromatous condition of the aorta itself, but there was little, and the mouths of both coronary arteries were perfectly free. On opening the coronary arteries along their full extent, the left one was found to be healthy, but the mouths of all the branches which opened out of the right coronary artery were nearly occluded by atheroma. In such a condition almost the only thing that one could have done was to have kept the patient perfectly at rest, and possibly digitalis, caffeine, and nux vomica might have helped to stimulate the right ventricle to increased contraction. Iodide of potassium might have been given, with the idea of causing absorption of the atheromatous material, but practically the complete rest, with massage to keep up the circulation and maintain the appetite, would have been the only treatment likely to do much good.

LAUDER BRUNTON.

DISEASES OF THE ARTERIES.

In considering the blood vascular system, it is necessary to bear in mind that the calibre of the arteries and capillaries is influenced by a variety of causes, the result being that they may be either unduly contracted or unduly dilated, and that these conditions respectively modify the amount of blood in parts, and affect the blood pressure throughout the arterial system generally. The causes determining these changes may have their origin in conditions of the blood which directly affect the vessel wall, or which operate through the nervous system, while in other cases the cause lies in an initial disturbance of the nerve centres themselves.

Contraction and dilatation are seen in their most simple form as the result of the influence of external agents, as cold and heat, which respectively produce contraction and dilatation, as indicated by pallor or redness of the exposed part. On the side of the nervous system, the first factor to note is the influence of the emotions in producing through the vasomotor nerves a corresponding pallor or redness, no doubt showing itself mainly in the face, but by no means necessarily or always confined to it. The influence of various organic and inorganic substances in influencing the calibre of vessels may here be mentioned, so that this factor may not be lost sight of. The significance of this latter is that in practical medicine we have certainly to deal with conditions in which deleterious or toxic substances are present in the blood, and influence in one or other direction the tonicity of the vessels. The state of the pulse in the infective fevers is sufficient illustration of what is here indicated. In them the tonicity of the arterial wall is an important guide in forming an estimate of the circulation, and relaxation is probably caused more by the toxæmia than by the mere elevation of temperature. On the other hand, in renal conditions where the state of the kidneys is such as to prevent efficient removal of the waste products produced during the physiological activity of organs and tissues, the arteries, and possibly the capillaries, become contracted, the result, presumably, of irritation by the retained excrement.

This whole question will be considered in the chapter dealing with the pulse, and reference is made to it here merely to bring it into line with other changes in the vessels which have to be considered in this section.

ABNORMALITIES.

It is necessary to bear in mind that abnormalities both in size and in distribution of the arteries occur. This is more especially necessary in connection with the radial arteries, for it not infrequently happens that a pulse is regarded as small and weak when the simple fact is that the vessel is abnormally small, or that the vessels at the lower part of the forearm are abnormally distributed. To avoid this error, both radials ought to be felt, and if they are both small, further investigation should be made into the anatomical arrangements. This can be done by tracing the vessel further up the forearm, or by examining the brachial artery, and ascertaining if there exists a due proportion between the pulse volume in the two vessels.

HYPERTROPHY.

Hypertrophy is the term applied to the enlargement which occurs in the branches of a collateral circulation, when the main channel has been occluded by disease, or by the surgeon on account of disease.

The term, however, is also applied to arteries in which the muscular coat—tunica media—is thickened. The condition is common, and is usually associated with the chronic forms of nephritis, but it may be present, although rarely, without this association.

The diagnosis is based upon the thickened state of the arterial wall—the radial being the test artery in all these conditions. The vessel is hard, and can be rolled under the finger; it has been long compared to whipcord, and the comparison is not unsuitable. The thickness is uniform, not in segments, and it necessarily varies within considerable limits in individual cases.

ARTERIO-SCLEROSIS.

The term arterio-sclerosis, often termed endarteritis deformans, has recently come into general use, but different physicians apply the term differently, frequently somewhat vaguely, and often inaccurately. The term is applicable clinically to thickening of arterial walls, as that condition manifests itself in the radial arteries and other vessels readily accessible to investigation.

Etiology.—Various views are held as to the state of the arteries in sclerosis, and varied interpretations have been offered of the changes noted. The experience of the writer of this article may be briefly indicated. Arterio-sclerosis occurs at different periods of life, usually after 40 years of age, but it is most commonly found in persons of 60 or more years. The etiology of the condition is open to question, but the writer has seen typical cases due to syphilis, others probably due to alcoholism, and others where the cause could not be satisfactorily traced. The presumption is that the initial fault is a toxæmia, the toxic factor being different in different cases.

Morbid anatomy.—The anatomical change is thickening of the arterial wall. The thickening is most marked in the tunica media, and consists, as a rule, of a hyperplasia of its muscle fibres; the intima shows a variable degree of thickening, due to hyperplasia of its fibrous elements; the tunica adventitia is also often thickened from hyperplasia of its fibrous tissue. With this thickening of the wall the lumen is, as a rule, diminished. The condition has atheroma sometimes associated with it; but many long stretches of arteries, such as an entire radial, may be examined without any considerable atheromatous change being found. The combination has, however, to be borne in mind, for with atheroma superadded there is a greater tendency to cerebral hæmorrhage.

The relation between this condition of the arteries and granular kidney is widely misunderstood. In arterio-sclerosis, such as has been indicated here, the renal arteries and their branches are similarly affected, and this gives rise to areas of arterio-sclerotic atrophy in the kidney, and to the naked-eye and microscopical characters of one form of granular kidney. On the other hand, a primary kidney cirrhosis ultimately produces changes in the systemic arteries, closely resembling primary arterio-sclerosis.

Symptoms.—The change is indicated by the thickened, and sometimes tortuous, condition of the wall of the radial artery. The blood pressure within this thickened vessel may be high or low. The symptoms which bring the patient under observation are very varied; they may be referred to the digestive system; they may be referable to the nervous system, causing complaint of insomnia, drowsiness, lethargy, muscular weakness, impaired memory, inability for mental concentration, and, in still more severe cases, local paresis or monoplegia. There is usually enlargement of the heart. The condition of the urine varies; it may be pale in colour, of low specific gravity, and there may be a history of frequent micturition, or of calls during the night to empty the bladder. At other times the same patient will pass a smaller quantity of dark-coloured urine, with a specific gravity of 1020 or more. In the majority of cases there is no albumin demonstrable by the ordinary tests; but in some cases albumin is present. The changes in the arteries are to be found in others besides the radials.

In the purer cases of arterio-sclerosis there is a progressive deterioration in the vital functions generally—the mental powers become weak, the circulatory and digestive powers enfeebled, and the muscular power diminished. These and all the other symptoms which may be present are due to diminished arterial blood supply to organs, to enfeebled metabolism, and to inefficient removal of waste products.

Diagnosis.—The thickening is to be distinguished from atheroma in this, that the thickening is uniform, that is to say, the vessel is thickened in its entire circumference and along its entire course. To the finger it feels like whip-cord or a thick-walled rubber tube. In atheroma the thickening is irregular, always more marked on one side of the vessel than the other, and it does not affect a vessel uniformly along any considerable length. In advanced atheroma the areas of calcareous infiltration are readily distinguished from the other parts.

Treatment.—This must be based on physiological principles. The basis is the regulation of diet, which requires to be just sufficient for physiological wants, easily digestible, and sparingly nitrogenous. Elimination of the waste products of metabolism is to be favoured by attending to the state of the skin, by keeping the bowels acting freely without purgation, and by the free use of bland liquids to ensure free urinary excretion. As regards medicinal treatment, iodide of potassium is useful in all arterial changes traceable to syphilis. Where blood pressure is low, cardiac and general tonics are indicated. On the other hand, if blood pressure be high, the nitrites are indicated, or even a mercurial laxative may be required.

ATHEROMA.

Etiology.—The precise causes determining the changes are not fully known, but gout, rheumatism, alcoholism, and syphilis are regarded as factors favouring their development. A tendency to this form of degeneration may be inherited, this being indicated by its appearance in comparatively early life. It is commonly regarded as a constant change in senility; and atheroma, more or less extensive, is almost invariably found in the arteries of old people. So much is this the case, that the old aphorism, "A man is as old as his arteries," is an axiom in practical medicine of far-reaching significance.

Morbid anatomy.—Atheroma is the most common abnormal condition which occurs in the arterial system. It affects the vessels irregularly, forming patches on the inner surface. The patches are raised, and whitish or yellowish in colour. In the small arteries the atheromatous patches are distinguishable from the outside of the vessel, owing to the opacity and thickening of the affected parts. On transverse section the artery usually shows thickening on one side only; the section has thus the "signet-ring" shape, the term commonly used to describe it. It is essentially an affection of the tunica intima, and is characterised by a thickening of this coat, the result of a hyperplasia of the subendothelial connective tissues. Along with this thickening, the deeper part, that is the part next the media, is the seat of fatty or atheromatous degeneration, and this area of degeneration becomes sooner or later more or less infiltrated with calcareous salts. The salts may be deposited so as to form small nodules, or, as in the large arteries, plates of considerable size. The large plates necessarily only occur in the large arteries, and of these the arch of the aorta is usually the part where the calcareous change is most advanced, although any or all of its large branches may be extensively affected. In the aorta the arch has sometimes an almost complete casing of this kind, so that slight violence leads to its cracking, and to laceration of the tunica intima. Instead of becoming infiltrated with lime salts, the area of fatty degeneration may extend so as to constitute what is known as an atheromatous cyst, the wall of which may give way, discharging its contents into the blood stream, and leaving an atheromatous ulcer. As regards the relation the changes bear to each other, the thickening of the intima may precede the atheromatous or fatty change; while at other parts the conditions seem to appear simultaneously, and to advance hand in hand. The condition leads ultimately to atrophy or to fibroid degeneration of the middle or muscular coat, and thus to lessening of the elasticity and the contractile power of the vessel.

Results.—The results of these changes in the arteries include some of the most common conditions met with in practical medicine, and it will be convenient to consider them under the headings of—(*a*) small arteries, (*b*) medium-sized, and (*c*) large arteries.

Small arteries.—Among the small arteries, the implication of the cerebral and the coronary vessels is of the greatest clinical importance.

The cerebral arteries occupy the foremost place in this relation. Owing to the physical and physiological conditions of the intracranial circulation, they are very frequently affected in this way. The larger arteries at the base, and their smaller nutritive branches, may all be extensively affected. Aneurysms may form upon any of them, and may attain sufficient dimensions to behave in many respects like a tumour of entirely new growth, and give rise to symptoms comparable to or identical with such. Other two results, however, may follow upon this affection of the arteries in this region, which are of still greater importance. The first of these is rupture and hæmorrhage; the second is thrombosis. The symptoms referable to each of these will be considered under Cerebral Lesions.

The coronary arteries are also frequently extensively and profoundly affected. They, however, do not manifest the tendency to rupture which is such an outstanding characteristic in the cerebral arteries; but, from the interference with their lumen, and with their elasticity, the blood stream passing through them to the heart muscle is impeded, and the heart muscle suffers from malnutrition. They are also not infrequently the seat of thrombosis, when, of course, the nutrition of the heart muscle is still further impaired.

Medium-sized arteries.—In the medium-sized arteries of the limbs, atheroma alone, or combined with arterio-sclerosis, may lead by thrombosis to cutting off of the arterial blood supply to whole or part of a limb. This is of pretty frequent occurrence in old people with diseased arteries, and is the cause of senile gangrene.

Large arteries.—In the large arteries atheroma leads to such great changes in their walls that the term *arteritis deformans* is applied to it. It is, however, only in the arch of the aorta, and in the large vessels arising from it, that the changes call for detailed consideration here.

Taking the arch of the aorta first; it may be affected in very different degrees. It may only show one or two small patches of no clinical significance. Even small patches, however, acquire importance if they are so placed as to encroach upon the lumen of the coronary arteries where they open from the aorta. When the arch is extensively affected, it becomes dilated in proportion to the amount of atheroma present, and the dilatation is most marked where secondary changes have followed upon the primary. The secondary changes which lead to dilatation are atrophy or fibrosis of the middle coat; either of these so weakening the wall of the vessel that it yields before the intra-arterial pressure. This may lead to dilatation of any of the anatomical segments of the arch, the ascending, transverse, or descending portion; but it may somewhat uniformly affect the whole arch or a large part of it, and produce what is known as an aneurysmal dilatation.

This aneurysmal dilatation gives various indications of its presence. The arch may rise so high that its pulsations may be visible in the episternal notch. The finger pressed into the notch readily feels the arch in these circumstances. In extreme cases there may be pulsation, appreciable to the hand placed on the upper part of the sternum and the adjoining cartilages and spaces. The area of percussion dulness in the same region, corresponds with the degree of dilatation. On auscultation, a systolic murmur may be present, and the second aortic sound is accentuated.

The arch may be extensively infiltrated with calcareous material, following upon atheroma, so that it may be transformed into an almost uniformly rigid tube; the vessel in these circumstances being always much dilated. When the aorta is extensively affected, the aortic cusps are also commonly affected, but this is not invariably the case. On the other hand, with but comparatively slight affection of the vessel, the valves may be profoundly altered in structure and in function. Whenever the arch is materially affected, its elasticity is correspondingly lessened, and as the normal elasticity is an important factor in facilitating the onward passage of the blood, the converse holds when the vessel is atheromatous or calcareous.

Diagnosis.—The diagnosis of atheroma depends upon the existence of changes in the radial arteries, and the occurrence of symptoms referable to intracranial hæmorrhage or thrombosis on the one hand, or of cardiac failure and degeneration on the other. These will be considered fully in the chapters on Cerebral and Cardiac Lesions. Atheroma of the radial arteries is recognised by the thickened and hard condition of the vessel wall; this not affecting it uniformly, but irregularly. When calcareous infiltration has occurred, the areas so affected are recognised by the rigid state of the vessel wall. While it may be legitimately inferred from this condition of the radial that the cerebral and coronary, as well as the large arteries, are similarly affected, it is not an absolute indication that these are equally diseased. Moreover, marked atheroma of the aorta or of the cerebral or coronary arteries is frequently found, without much evidence of the condition being present in the radials. Still, while allowing for these comparatively exceptional cases, the state of the radials may be taken as a fairly reliable indication of the condition of the arterial walls in other parts.

ENDARTERITIS OBLITERANS.

This condition differs essentially from atheroma. It is characterised by a thickening of the tunica intima, due to a hyperplasia of the subendothelial fibrous tissue. It affects the intima uniformly and progressively, so that the lumen of the part of the vessel affected becomes gradually smaller, until it is finally obliterated. The thickened part does not undergo the fatty and atheromatous degeneration which characterises atheroma, neither does it become infiltrated with lime salts. Occurring in the vessels of the brain, it is due to syphilis; in fact, when due to syphilis, the cerebral vessels are those which are, if not solely, certainly mainly affected. The condition does not affect uniformly any extensive tract of artery, but only circumscribed portions of it, giving rise to nodular thickenings and opacities in its course. It leads to a gradual diminution of blood supply to the portions of brain supplied by the affected vessel, and to consequent cerebral degeneration, until, finally, the supply is completely cut off. Before the endarteritis completely occludes the vessel, it may be blocked by thrombosis, when, of course, the consequences to the brain are the same as in the former case. Although endarteritis obliterans is, in its more important manifestations, syphilitic in origin, a corresponding anatomical change occurs in the kidneys and lungs in cirrhosis of these organs, and not infrequently in areas of carcinomatous new growths.

AORTITIS, OR ACUTE ARTERITIS.

This condition is analogous to syphilitic endarteritis obliterans. It specially occurs in the aorta, and is characterised by the presence of raised grey swellings of considerable extent on the inner aspect of the vessel. The condition may affect the greater part of the arch. The areas are due to a thickening of the tunica intima, corresponding anatomically to that present in atheroma and obliterative endarteritis; but there is little or no tendency to fatty degeneration, as in atheroma, and the wall is not uniformly affected, as in endarteritis obliterans affecting smaller arteries. In addition to the affection of the internal coat, both the middle and outer coats show evidence of inflammation, by the occurrence of areas of small-celled infiltration in them. Similar areas are also found in the thickened intima. The condition is probably syphilitic. As regards diagnosis, it has not yet been clinically differentiated from atheroma.

CALCAREOUS INFILTRATION.

In addition to the calcareous infiltration of arteries, which occurs in atheroma, another form occurs in which the infiltration is into the middle coat of the vessel. The condition is met with affecting extensive tracts of middle-sized and smaller arteries, both those carrying blood to the viscera or supplying the limbs. The vessels affected are converted into rigid tubes through which the blood continues to flow, although in a sluggish stream. The condition is diagnosed by the state of the arteries within reach of digital examination.

MILIARY ANEURYSMS.

These are minute aneurysms which form on the nutrient branches of the cerebral arteries. They may be present in great numbers, and are a common site of small hæmorrhages in various parts of the brain. The occurrence of these hæmorrhages from time to time is marked by symptoms which are not infrequently mistaken for the "congestive attacks" of general paralysis. The aneurysms are associated with arteriosclerosis, and in some instances are almost certainly syphilitic. The minute changes which lead to their formation have not yet been satisfactorily determined, although Charcot and Bouchard regarded them as following upon a periarteritis, while Zenker placed the initial change in the intima.

ANEURYSM.

Etiology and morbid anatomy.—An aneurysm is a circumscribed dilatation of an artery, and as the aorta is the commonest site of the lesion, we shall confine our description to a consideration of the symptoms which are caused by it in this vessel. Atheroma is certainly the commonest cause of aneurysm. That aneurysm is more commonly produced in the aorta than elsewhere, is due to the special strain to which its walls are subjected in physical strain of all kinds. This is borne out by the fact that it is more common in the male than in the female sex; and amongst males it occurs most frequently in men who follow laborious occupations and who require from time to time to put forth great physical effort. It occurs in labourers, iron-workers, soldiers, and others. Some authorities hold that aneurysm always has its origin in syphilis; but unless it be contended that atheroma is invariably syphilitic—a doctrine which cannot be accepted—the contention has always seemed to us to be unreasonable. The dilatation may form a distinct sac or cup-like diverticulum; or it may constitute merely a localised bulging. It may occur at any part of the arch, but it may involve the greater part of it in its entire circumference. In this case there is much deformity of the internal surface of the vessel, and the aortic cusps are often much altered. The sac may be largely composed of some of the arterial coats, but frequently a considerable part of it is of new formation. The interior has deposits of fibrin and other elements derived from the blood.

Symptoms and diagnosis.—The symptoms which are manifested depend upon the situation and the size of the aneurysm, and are subjective and objective. The subjective symptoms may be pain, palpitation, impairment of voice or of vision: but none of these are pathognomonic, or do more than suggest the possibility of aneurysm. The diagnosis depends on the existence of a variety of phenomena, and of considerations referred to below. The evidence is partly direct and in part indirect.

Inspection and palpation.—The presence or absence of visible pulsation necessarily depends upon the situation of the aneurysm. It can only be present when the aneurysm is so situated that it comes into contact with the chest wall, or extends upwards into the neck from behind the manubrium sterni. An aneurysm may thus visibly pulsate to the right or left of the upper part of the sternum, or above it. The pulsation also varies according to the size of the aneurysm; in large aneurysms, the upper part of the sternum, and the adjoining interspaces, cartilages, and ribs, show the pulsations of the underlying sac. The pulsation, when it is visible, is of course readily felt on palpation; but in some cases the transmitted pulse of the aneurysm may be felt when there is no visible pulsation. This is of importance as a diagnostic point, because of the absence of such a transmitted pulse from the normal aorta. In addition, the closure of the aortic valves may be markedly accentuated, and the resultant vibrations appreciated by palpation. When the aneurysm is large, there may be distinct bulging of the upper part of the sternum and adjoining parts. In comparatively small aneurysms, when sacculated, there may be distinct localised bulging, usually on one or other side of the sternum, or above the sternal notch, according to the part of the arch affected, or to which of the vessels arising from it is the seat of the aneurysm. In abdominal aneurysms the swelling and pulsation may be very marked.

Tracheal tugging is a sign which should be always looked for, as it is occasionally present in aneurysm of the arch of the aorta. The name is applied to a rhythmic drawing down or tugging upon the larynx, the result of the aneurysmal sac having formed strong adhesions to the trachea. It is best felt by raising the chin and grasping the thyroid cartilage between the thumb and forefinger, and pulling it gently upwards.

Percussion.—The percussion sound over the aneurysm is dull. It is also dull over other tumours and over various lesions occurring in the upper part of the anterior edges of the lungs. Bearing these facts in mind, the existence of an area of dulness in a circle the diameter of which would traverse the sternum about the level of the second cartilage, might be a sign of aneurysm. Without the presence of other and more important signs, this is, however, of little value.

Auscultation.—On auscultation there may be a murmur which is systolic in time, that is, which coincides with ventricular systole. The murmur is the result of fluid veins being formed by the blood passing into the wider channel of the aneurysm. A murmur is, however, not present in all cases of aortic aneurysm, and it has to be borne in mind that a systolic murmur heard over an aneurysm may be produced not in the aneurysm itself, but at the aortic orifice, the result of a lesion there. The aortic orifice is also, no doubt, the seat of origin of a diastolic murmur which may be present over the aneurysm. At all events, if produced in the sac, it can hardly occur without an accompanying aortic reflux.

These are the signs which may be present on the physical examination of the part, but there are signs of another class which have to be inquired into, and which often materially assist the diagnosis. These signs are produced by pressure upon surrounding tissues and structures, and the structures so pressed upon are necessarily determined by the anatomical seat of the aneurysm.

Symptoms due to pressure.—The main structures, pressure on which gives rise to symptoms, are—bone, as the vertebræ, sternum, or ribs; large blood vessels, as the superior vena cava, the right or left innominate, and the pulmonary artery; nerves, as the phrenic, pneumogastric, recurrent laryngeal, or sympathetic; the œsophagus, or one of the hollow viscera of the abdomen; the trachea, or one or other bronchus.

Pressure on bone leads to erosion, and this is associated with pain. When the aneurysm is on a part of the vessel which is in contact with the vertebræ, pain in the back, between the shoulders, may be an early symptom. As the case advances, there is tenderness on pressure over the affected bones, and the erosion may become so advanced that even spinal curvature may result, and irritation may be set up in the spinal meninges, and may extend to the cord. On the other hand, when the aneurysm is so situated that its expansion is forwards, the sternum, ribs, and cartilages may be eroded, thinned, and bulged to a very great degree forwards. The bulging may indeed be carried to such an extent that the aneurysmal sac is covered by little save skin, which may be so irritated and stretched that it becomes red and inflamed.

Pressure on any of the large venous trunks, obstructing the flow of the blood through them, leads to congestion behind the point of obstruction; thus, if the pressure be on the superior cava, the vessels in the neck, face, and upper limbs become turgid. When the obstruction is great the congestion is proportionately great, and causes œdema of the parts mentioned. The vessels of the brain, of course, suffer with the others.

Among nerves, the nerve which is most commonly pressed upon is the left recurrent laryngeal, as it hooks round the aorta before proceeding upwards to its distribution to the muscles of the larynx. Pressure here causes paralysis of the left vocal cord and a corresponding affection of voice. The nerve on the right side is sometimes involved. The sympathetic is also not infrequently pressed upon, and leads to inequality of the pupils or to unilateral sweating of the face and neck. Symptoms due to pressure upon the phrenic or the pneumogastric are rare, but the diaphragm may be paralysed. It is also probable that involvement of the plexuses, to the formation of which the pneumogastric and sympathetic contribute, accounts for changes which sometimes occur in the lung, and which appear to be the result of trophic disturbances. The nerves passing out to the periphery may be implicated, causing sensory and motor disturbances.

Pressure on the œsophagus is common, and may interfere with swallowing, leading to dysphagia or even to complete obstruction. In all cases of œsophageal obstruction, the possibility of aneurysm being the cause has to be borne in mind, because of the danger of rupturing the aneurysm to which the patient would be exposed were an attempt made to force a bougie through the obstruction. In the abdomen, pressure on various structures produces manifold effects, even such a change as cutaneous bronzing having been seen.

Pressure on the trachea at its lower part leads to dyspnœa in proportion to the amount of mechanical obstruction produced by the pressure. It also leads to irritation of the mucous membrane and sometimes to distinct stridor in respiration, with a brassy or croaking character of cough, which is quite characteristic of laryngeal obstruction. Pressure on a bronchus, like pressure on the trachea, produces more or less mechanical obstruction to the entrance of air into the corresponding lung, and to a proportionate dyspnœa, or to dyspnœa on exertion. The bronchus is, however, also irritated by the pressure, the mucous membrane becoming inflamed and catarrhal. This causes cough, and the expectoration, when present, consists of the secretion from the inflamed area. If the inflammation be intense, the expectoration may be more or less sanguineous or blood-stained. When the obstruction is considerable, changes occur in the lung which are the result of collapse and of chronic inflammatory changes, and are identical with those caused by pressure upon a bronchus from other causes. These changes and the physical signs produced by them may be the only evidence during life which may lead either to the diagnosis or the suspicion of aneurysm. The changes which occur in the lung, while probably in the main the result of obstruction to the free entrance and exit of air, appear in some cases to be in part the result of trophic changes, brought about by pressure on nerve filaments belonging to the nerve plexuses in this situation.

Two other points connected with the circulation have yet to be indicated. The first of these is the comparison of the two radial arteries. There is not infrequently a decided loss in equality, the one being much smaller than the other; and sometimes there is even a slight difference in time in the pulse in the two vessels. The condition is due to the involvement of the parent artery, as it arises from the aortic arch, in such a manner as to partially obstruct its lumen and the flow of blood into it. The second is the displacement of the heart downwards and outwards when the aneurysm is large, and the hypertrophy of the left ventricle, which may be present even with a comparatively small aneurysm.

X-rays in the diagnosis of aneurysm.—The Röntgen rays may be the means of determining both the existence and the locality of internal aneurysm.

Course.—The further course of a case of aneurysm is frequently determined by the complications already referred to. Changes in the lung, obstruction of the œsophagus or trachea, and obstruction to the venous return to the heart, may induce progressive debility, and terminate in death from exhaustion and inanition. Rupture of the aneurysmal sac is, however, the more frequent mode of termination, death occurring from hæmorrhage, which may be external or internal. External rupture only occurs in those comparatively rare cases in which the aneurysm enlarges enormously forwards, and leads to erosion of the sternum or ribs, and finally to inflammation and ulceration of the skin. Internal rupture is much more common, and may occur in a variety of directions, namely, into the œsophagus, the trachea, the bronchi, the lung, the pleura, or the pericardium. In all these death speedily follows, if the hæmorrhage be large; but there may be several recurrences of slight hæmorrhage before the larger and fatal one occurs.

Occasionally an aneurysm opens into one of the large veins, as the innominate. This is evidenced during life, not only by the distension of veins, but by the presence of a wave in them synchronous with the distension of the aneurysm, and therefore with ventricular systole. An aneurysm at the commencement of the aorta may open into the pulmonary artery, which leads to an admixture of arterial and venous blood going to the lungs, and a corresponding deficiency in aeration, with its attendant consequences —cyanosis and dyspnœa. Further, it may rupture into one of the auricles, or even into one of the ventricles, the right being the more common.

Prognosis.—The prognosis of aneurysm depends upon its position and size, and upon the patient's circumstances. If enlargement is occurring, in spite of rest and suitable treatment, even the immediate prognosis is bad, for rupture may occur at any moment. Cure in the true sense is, of course, impossible for aortic aneurysm; but in many cases it is undesirable to take a gloomy view, for life may be prolonged for many years, and death occur from some intercurrent affection, neither directly nor indirectly attributable to the aneurysm.

Treatment.—The treatment of aneurysm has to be regarded both from the standpoint of prevention and of palliation. The cure of an established aneurysm, in the sense of restoring the vessel to the normal state, is impossible, and the term can be used only in a relative sense, or as a figure of speech.

The prevention of the lesion involves the consideration of the causes and possible treatment of atheroma. Some physicians contend that aneurysm is, in the great majority of cases, if not in all, the result of syphilis, which implies that the changes in the vessel preceding the development of aneurysm are syphilitic. This doctrine has not obtained universal acceptance; and there can, we think, be no doubt that atheroma occurs in rheumatic and gouty persons, or in persons whose tissue metabolism is modified or influenced by a diathesis in one or other of these directions. This being so, the aim of the physician who recognises the early indications of arterial degeneration must be to combat the diathesis, and thus to arrest or retard the progress of the condition. When degeneration is present, the avoidance of physical strain of all kinds, which increases intra-arterial pressure, is to be studiously avoided; for, under such strain, the degenerated and weakened arterial wall yields, and may do so suddenly and abruptly at some specially weak point.

When the aneurysm is established, the first consideration is a like avoidance of all causes which increase intravascular tension. With this object in view, arduous physical labour or exercise must be given up. This point must be insisted upon, if the enlargement of the aneurysm is to be prevented or delayed. It is, moreover, desirable that the patient should have an intelligent understanding of his own case, so that he may avoid all sudden temptations to physical strain. If there be a suspicion of syphilitic taint, treatment with the iodides is indicated. The general condition of the circulation ought to be regulated; if there be a tendency to plethora, or if there be evidence of general vascular degeneration, rich and stimulating foods of all kinds are to be avoided, and all alcoholic liquors, save in very small quantity. Particular attention should be directed to the state of the bowels, care being taken that they move regularly, fully, and without straining. This last precaution is liable to be overlooked, and it has to be constantly borne in mind that straining at stool is a form of effort which is dangerous, and requires special instructions for its avoidance.

When an aneurysmal sac is enlarging, or has attained a size which makes the possibility of the rupture a cause of anxiety, the question of further treatment has to be carefully considered. It is unnecessary to dwell in detail upon aneurysms in which the question of ligature arises. These are fully considered in works on surgery. Here it is only necessary to bear in mind that the principle underlying treatment by ligature is the arrestment of the blood stream through the sac, and thus the removal of the danger that the sac should give way before the blood pressure.

From the medical standpoint the aim is different. The pressure of the blood stream is moderated by rest, and by means of food and drugs. These have also supplementary beneficial action in favouring the precipitation of fibrin within the sac, which is thereby mechanically strengthened and saved from the direct intravascular pressure. Tufnell's treatment was directed to facilitate and hasten this intra-aneurysmal coagulation. It consists in keeping the patient absolutely at rest in bed, and diminishing the diet to such a degree that it is only sufficient for the bare support of life. The treatment got an extensive trial after it was first advocated, but the results have not been sufficiently satisfactory to have led to the general adoption of a method which is exceedingly irksome, and even intolerable to many patients.

With a similar object in view, Balfour advocated the administration of iodide of potassium. This treatment is as satisfactory as any other. We believe, however, that the amelioration of symptoms, and any reduction in the size of the sac, are the result of rest and the regulation of the blood pressure rather than anything else. The power of iodide of potassium to reduce blood pressure must, however, have full weight given to it, and its administration, combined with rest and a careful dietary, may be adopted with advantage. It requires, however, to be carefully watched and regulated. The iodide serves another useful purpose in relieving the pains which are liable to occur in the course of the affection. In advanced cases, especially where bone is being eroded by pressure, the iodides may require to be combined with morphine, or the latter may have to be administered hypodermically. More recently the hypodermic administration of gelatin dissolved in artificial serum has been employed. It is as yet too soon to express any opinion as to its value.

One or two other resources remain for consideration in advanced cases, where the aneurysm bulges to such an extent on the surface that external rupture appears to be imminent. One of these is galvano-puncture. The object of galvano-puncture is to induce coagulation, and thus to protect the wall of the aneurysm from the direct distensile effect of the intravascular pressure; this, as has been already pointed out, being the object aimed at in other methods of treatment. It is well to point out here that, in the nature of things, it is only to sacculated aneurysms that this method of treatment is applicable. In diffuse or localised aneurysmal dilatation, the formation of clot is not suitable, and would be dangerous on account of the ease with which the clot could be detached and lead to embolism. In cases where it is desired to use all possible means of inducing coagulation, galvano-puncture may be aided by the introduction of fine gold wire into the sac. Another method is that introduced by Macewen, scratching the interior of the wall of the sac so as to roughen it and favour coagulation.

THROMBOSIS AND EMBOLISM.

Thrombosis is the coagulation of blood in a vessel. It has already been referred to under Atheroma and Obliterative Endarteritis, these being the conditions in which it mainly occurs. It is of especial gravity when occurring in the cerebral or coronary arteries. It, however, also occurs in the arteries of the limbs, giving rise to *senile gangrene*, from the cutting off of the blood supply to the part.

Embolism is the conveyance and arrest of some foreign body in an artery. It usually consists of a portion of detached fibrin from endocarditis, or from an aneurysm, and occurs at a point where the vessel bifurcates. It commonly occurs in internal organs, as the brain, spleen, and kidney, and gives rise to symptoms which are considered under these organs. If the embolus be infective, an acute inflammation of the arterial wall ensues, which extends to surrounding tissues, and usually terminates in abscess formation.

WILLIAM RUSSELL.

DISEASES OF THE VEINS.

DISTENSION.

As a result of the anatomical structure of the wall of veins, they are capable of a degree of distension that is not possible in the arteries. This distension occurs on the distal side of any point at which obstruction has occurred. Cardiac disease and pulmonary disease are the common causes of engorgement and distension of the whole venous system; cirrhosis of the liver is the common cause of a similar distension of the portal system. Apart from these, more localised distensions occur from obstruction in the course of one of the principal veins, either from pressure on it by tumour, or from conditions of the vein wall which lead to complete thrombosis, or inflammatory exudation. When obstruction occurs, the branches normally returning their blood by the occluded vessel become engorged, distended, and elongated. The result is lividity and œdema of the part. Death of the part does not usually occur, as the collateral venous circulation is generally sufficient to prevent this.

VARICOSITY.

This is a condition of localised and more or less permanent distension of veins. The condition is most frequently seen in the lower limbs, and there it is the result of prolonged standing in persons who presumably have abnormally weak vein walls. The tendency is in some instances distinctly hereditary. As a result of prolonged over-distension, the vein valves become incompetent, and this necessarily leads to a still higher degree of distension, which becomes a source of danger from the ease with which at any part a vessel may give way from slight local injury. When an accident of the kind occurs, there is very profuse hæmorrhage, which not infrequently has a fatal issue, from the ignorance of the person to whom the accident happens as to how the bleeding may be arrested.

A limb whose vessels are varicose is also liable to develop ulcers from slight injuries; the reason being that the soft tissues are poorly nourished owing to the obstruction to the circulation, and that consequently both their power of resistance and their reparative powers are lowered.

Varicosity is, however, frequently the result of pressure upon a venous trunk. The most common cause of this is pelvic tumour, which compresses one or both iliac veins and leads thus to varicose veins in the lower limbs. Pelvic inflammation, by surrounding and compressing the iliac or its external branch, may produce a like result.

Varicosity also occurs in venous radicles and capillaries on the face (especially the cheeks and nose), on the ears, and on the posterior fauces. On the face and ears the condition is induced or favoured by a variety of causes—as mitral lesion, pulmonary disease, exposure, alcoholism, gout, and senility. In the fauces it results from chronic or recurring pharyngitis, and may be gouty.

NEW GROWTHS.

New growths, with the exception of tubercle, do not develop primarily in intimate connection with veins. Veins, however, are frequently secondarily affected, this occurring mainly by extension from adjoining parts. In this way the wall of a vein may be the seat of a cancerous, sarcomatous, or tuberculous lesion. This may lead to thrombosis at the affected part, and consequent occlusion of the channel. It may, however, lead to ulceration, with escape into the blood stream of tuberculous, cancerous, or sarcomatous material, which is then carried to other parts or organs, leading to secondary infection of these.

PHLEBITIS.

Veins are frequently the seat of inflammation. It may either occur as a direct extension from adjoining wounds or tissues; it may be primary, or it may be dependent upon causes to which reference will presently be made. It is characterised anatomically by distension of the vaso-vasorum and exudation into the vein wall. The affection of the vessel wall leads to precipitation of fibrin on its inner surface, and usually to complete thrombosis.

Etiology.—Phlebitis may be idiopathic. It sometimes occurs without any apparent local cause, in rheumatic or gouty subjects, and the writer has met with cases in the popliteal vein, which have been erroneously diagnosed as rheumatism. It is also liable to occur in the course, or during convalescence from, typhoid fever. It is not uncommon in the later stages of pulmonary phthisis, and it is then of very grave significance. It is liable to occur after childbirth, not only in the uterine and pelvic veins, but also in those of the lower limbs. In these three conditions, the inflammation is almost certainly due to micro-organismal infection, but while this is the case they are by no means of equally grave significance. When the inflammation is septic, and goes on to suppuration, the condition is necessarily much more grave, for the septic débris of the broken-down clot is carried by the blood stream, and gives rise to secondary foci of inflammation wherever arrested. This, however, only occurs in the more highly infective cases, which are comparatively rare.

Symptoms and diagnosis.—The condition is recognised clinically by pain at the seat of the inflammation and tenderness on pressure. If the affected vein be superficial, there is redness around the vein, extending to some distance from it. When thrombosis has occurred, the vein, if within reach of the finger, is hard and firm, as well as tender. When a large venous trunk is thrombosed, the venous return is impeded; the branches on the distal side of the obstruction are distended, and there is œdema of the part. The pain, tenderness, distended veins, and œdema are well illustrated in phlebitis of the femoral or popliteal vein; their cord-like hardness can also frequently be made out. Other large trunks may be affected, such as the humeral, iliacs, mesenteric, and even the portal. In phlebitis of the last of these, ascites results.

Prognosis.—In idiopathic, rheumatic, or gouty cases, the prognosis is good; the inflammation subsiding, the clot disintegrating, and the lumen of the vessel being completely restored, although this may be effected somewhat slowly. In the purulent form the prognosis is more grave, and the degree of gravity depends upon the initial cause.

Treatment.—In ordinary cases the treatment consists in giving absolute rest to the part, and to ensure this it is necessary to keep the patient in bed for some time at least. Pain is relieved by anodyne fomentations, my favourite remedy being the application of equal parts of extract of belladonna and glycerin, with hot poultices over that. When the inflammation has subsided, and œdema remains, or comes on when the limb is allowed to hang down, a firm bandage, preferably of rubber, is of advantage. Of remedies to be given internally, potassium iodide is the most useful. When the phlebitis is purulent, the foregoing treatment is applicable until the question of surgical interference presents itself.

WILLIAM RUSSELL.

DISEASES OF THE CAPILLARIES.

There are only few affections of the capillaries to which reference need be made. Engorgement occurs from causes which lead to distension of the whole venous system, as heart and lung affections. It is characterised clinically by a bluish hue of exposed parts, such as the face and hands, and is known as cyanosis. Fibrosis of the arterial capillaries has been described in Renal Cirrhosis, the kindey change being regarded by some as part of a more diffuse affection. Fatty degeneration occurs in anæmia, more especially in pernicious anæmia, where it is a cause of capillary hæmorrhage into the retina, and occasionally from mucous and serous surfaces.- It also occurs in phosphorus and other forms of poisoning.

A capillary pulse is seen in cases of marked aortic regurgitation. It is brought out by drawing the finger nail rapidly across the forehead, when an alternate flush and pallor becomes perceptible in the tract traced by the nail.

Spasm of the arterial capillaries, with complete anæmia of the part and consequent local death, occurs as a symmetrical lesion on both hands or both feet in Raynaud's disease.

WILLIAM RUSSELL.

DISEASES OF THE LYMPHATICS.

Distension of lymphatics occurs from occlusion of large branches. This occurs in the tributaries of the thoracic duct when from any cause it is occluded. In this case the contents of the distended lymph vessels escape into the abdominal cavity, and give rise to chylous ascites. A corresponding condition, due to obstruction of lymphatics in the genito-urinary tract, by the ova of *Filaria sanguinis hominis*, produces chyluria. Lymph scrotum and elephantiasis result from blocking of the main lymph channels from the affected parts, by the same parasite or its ova. Localised distension occurs in the tongue, giving rise to a condition known as macroglossia.

Owing to the anatomical connections of the lymphatic system, its radicals are readily affected secondarily, from primary centres of disease with which they are connected. This is strikingly evidenced in septic inflammations of external parts, where, extending from the primary site of the inflammation, the lines of inflamed and red lymphatics are quite manifest. The inflamed lymphatics are painful and tender.

It is only necessary, further, under this head, to bear in mind that septic infections extend by lymphatic channels, in connection with internal as well as external foci; and that infective matter is carried by them to distant lymph glands, or to other organs. This is well seen in tuberculosis, and in malignant disease, both of sarcomatous and cancerous nature.

WILLIAM RUSSELL.

SECTION VII.

RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

Affections of the nasal passages comprise a very numerous group. For the detection of these the methods of examination known as anterior and posterior rhinoscopy are employed. Anterior rhinoscopy, or the use of the nasal speculum, enables the examiner to see the mucous passages, septum, inferior turbinated body, and parts of the middle and superior turbinals. For posterior rhinoscopy, a rhinoscopic mirror and tongue depressor are required. The rhinoscope is introduced behind the soft palate, and, by manipulation of the mirror, the observer should make out, in health, the posterior aspects of the uvula and velum palati, the septum, the turbinated bodies within the fossæ, Eustachian tube, fossa of Rosenmüller, and pharyngeal tonsil.

RHINITIS.

Acute Rhinitis.

An acute simple catarrh of the nasal mucous membrane.

Etiology.—Coryza is most commonly ascribed to exposure to cold and draughts of air, especially when the surface of the body is heated. Delicate persons are prone to be attacked, and there are some individuals who seem to have a natural predisposition to the malady. It is undoubtedly infectious, and some atmospheric conditions seem to favour its occurrence, but the nature of the *contagium* has not as yet been made out. Acute rhinitis is also frequently associated with certain specific fevers, notably measles, and it is produced by certain drugs, more particularly iodide of potassium, when given in small doses.

Morbid anatomy and pathology.—The mucous membrane is swollen from congestion and exudation. The serous exudation is unusually abundant, in great part owing to the structure of the membrane, which contains serous and mucous glands. The epithelium proliferates and leucocytes are latterly found in the exudation. Numerous forms of bacteria have been described in the secretion—the *pneumococcus, streptococcus pyogenes,* and others—but their precise rôle is not decided.

Symptoms.—There is slight pyrexia, accompanied by a feeling of malaise. Constipation is generally present. An irritating stuffiness of the nostrils, frequent sneezing, and the discharge of a watery serum, constitute the nasal symptoms. The pharynx is injected and slightly swollen; the larynx is often congested, causing huskiness of the voice, and the neck feels stiff. A feeling of fulness, or heaviness, or actual pain in the frontal region, due to congestion, is generally experienced. The conjunctivæ are also involved in the congestive process, giving rise to irritation and "watering" of the eyes. Sometimes the catarrh extends to one or other or both Eustachian tubes, and a degree of deafness, with tinnitus, is produced. In infants, suffocative symptoms may arise. As the attack subsides, the nasal discharge becomes scanty and muco-purulent.

Treatment.—Endeavours are often made to cut short or abort the attack, and for this purpose opium or morphine is given at the commencement. But a better plan is the popular one of inducing diaphoresis on retiring to rest. Topical applications, such as menthol and weak solutions of cocaine, are often used to allay the symptoms. Children sometimes suffer severely, when steam inhalations should be resorted to: and to reduce the swelling of the mucosa, a 5 per cent. solution of cocaine may be applied with a brush. An obstinate coryza is often quickly relieved by a change of air.

CHRONIC PURULENT RHINITIS.

An ordinary coryza, by frequent recurrence, may lay the foundation of a chronic affection; but there are generally constitutional causes at work in such cases. Sometimes a purulent form of rhinitis occurs in the newly-born, from gonorrhœal or leucorrhœal infection, while in adults many specific purulent discharges from the nose are met with. The treatment of such cases will be considered in connection with the conditions of hypertrophic and atrophic rhinitis.

CHRONIC HYPERTROPHIC RHINITIS.

A chronic inflammation of the nasal mucous membrane, accompanied by cellular proliferation in the deeper parts, and hypertrophy of the constituent tissues generally.

Etiology.—Chronic hypertrophic rhinitis is found in persons who, from the nature of their occupation, or from some defect in the nasal passages, are liable to attacks of acute rhinitis, from which they recover but imperfectly. The inhalation of an irritant atmosphere is thus a fertile source of the disease. Certain constitutional conditions, *e.g.* obstruction of the circulation, and hepatic and gastric disorder, also favour the chronic congestion of the membrane.

Morbid anatomy.—There is an irregular thickening of the mucosa from the growth of granulation-like tissue and hyperplasia of individual structures. Spurs and outgrowths are thus formed. There is a symmetrical hypertrophy of the anterior and posterior turbinated bodies, with deviation of the septum; or polypoid tumours may occur on the anterior or posterior extremities of the inferior turbinal. Congestion and hypertrophy are also found in the adenoid structures of the naso-pharynx.

Symptoms.—The obstruction in the nasal passages gives rise to mouth-breathing, with its attendant phenomena. The patient is frequently blowing the nose, and there is considerable annoying discharge of muco-purulent matter, sometimes tinged with blood, especially if much effort is used to get rid of secretion. Attacks of sneezing are common, accompanied by acute exacerbations of the swelling of the membrane. Smell, taste, and hearing are often much impaired.

Diagnosis and prognosis.—Hypertrophic rhinitis is a more or less generalised affection, and is thus distinguished from new growths, which are localised and of a recognised consistence in many cases. The rhinitis characteristic of certain neuroses, particularly hay fever, has a different history, and the engorged membrane is reducible by cocaine. An improvement can always be brought about, though the disease is one requiring considerable time and care. In some instances a cure may be effected.

Treatment. — In chronic rhinitis generally, including purulent rhinitis, cleansing solutions should be freely used to the interior of the nares. These may be employed twice or thrice daily in the form of a spray, or douche, or by means of a syringe. The douche should be used with caution. Alkaline and antiseptic solutions, such as those containing borax, carbolic acid, chlorate of potassium, and "listerine," are best. Insufflations of finely powdered silver nitrate (with starch) or alum are also used. When there is much thickening of tissue, the best plan probably consists in scoring the surface with the galvano-cautery, though such caustics as chromic acid and glacial acetic acid are also used. Actual outgrowths, such as polypi, require removal by the snare. In all such cases, cocaine must be thoroughly applied in the first instance, and, after operation, the nostril should be plugged with cotton-wool, impregnated with an antiseptic ointment, such as eucalyptus and vaseline. The naso-pharynx should always be examined for adenoid growths. Constitutional treatment, with a view of improving the general nutrition and tone, may be indicated in many cases.

HAY FEVER.

In this neurotic affection we find a special form of hypertrophic rhinitis of vasomotor origin. There is a peculiar morbid sensibility of both the nasal and conjunctival mucous membranes to the action of certain substances present in the atmosphere, notably the pollen of grasses. As a consequence, the nasal mucosa, and particularly the cavernous tissue over the inferior turbinated body, becomes much swollen, and secretes an abundant watery fluid. A careful examination should be made of the interior of the nose. Thickening over the turbinals should be freely scored with the galvano-cautery. This is probably the most efficacious treatment, though a vast number of other means are advocated, such as swabbing the nostrils with glycerinated solutions of corrosive sublimate or carbolic acid, with solutions of menthol, cocaine, etc. Opium and other drugs have been administered internally, with uncertain results. See under "Bronchial Asthma."

CHRONIC ATROPHIC RHINITIS—OZÆNA.

A widespread atrophic degeneration of the mucous membrane of the nose, accompanied by a low type of chronic inflammation, giving rise to a purulent discharge and the formation of adherent crusts having a peculiarly offensive odour.

Etiology.—Ozæna is met with chiefly in females, coming on in childhood or about the period of puberty, and rarely develops after the age of 20. As a symptom it is met with in ulcerative affections, especially necrosis of the nasal bones. Chlorosis and kindred conditions of vitiated blood predispose to the disease. Sometimes a hereditary tendency is observed.

Morbid anatomy and pathology.—In many cases the disease begins with inflammatory swelling of the mucosa and purulent discharge. In other instances the inflammatory manifestations are accompanied by wasting and fatty degeneration of the various epithelial structures of the membrane, *ab initio.* The glands in particular are the seat of the fatty degeneration. Some cases of ozæna have been attributed to obstruction in the passages, causing retention and decomposition of secretion. The peculiar stench characteristic of the disease is considered due, in part at least, to the presence of a bacillus termed the *B. fœtidus.* Other micro-organisms have been found in ozæna, but their significance has not been determined. Most of them are probably contingent on the disease. When ozæna is at all advanced, the turbinated bones are found to be much atrophied.

Symptoms.—The nostrils are dilated and encumbered with adherent crusts, which extend to the naso-pharynx, and emit a disgusting odour. These crusts cause irritation, which is often aggravated by a thin, purulent, and but scanty discharge. The pharynx is dry and irritable, and the trachea may become secondarily involved. The sense of smell is lost, and frontal headache is common. Eye and ear affections, usually of a catarrhal character, are not infrequently met with.

Prognosis and treatment.—Careful and systematic treatment will greatly mitigate the symptoms, but a cure is hardly to be expected. As time goes on, the disease tends to lessen in severity. Thorough local and general treatment should be adopted as soon as the disease makes its appearance. Various preparations of iron and arsenic, and particularly cod-liver oil, are most suitable for internal use. Locally, the crusts must be loosened by spraying or douching with such alkaline solutions as are indicated under Hypertrophic Rhinitis, and the cavities thoroughly cleansed. Paroleine containing eucalyptus or menthol in solution may then be introduced by the oil atomiser to control fœtor, or the nasal inhaler may be employed. In like manner ointments may be applied, after using the spray or douche; those of mercury, eucalyptus, and aristol being probably most serviceable.

INFECTIVE GRANULOMATA.

Tuberculosis of the nasal mucosa is a rare condition, but in the form of lupus we sometimes have a tuberculous affection, which may either be primary, or else an encroachment of this disease from the face. Lupus is attended by ulceration, which often eats into, and causes perforation of, the cartilaginous septum. It is distinguished from syphilis in its history, slow course, non-implication of bony structures, and in lack of response to specific treatment. Both constitutional and local measures, but especially the former, must be carefully and systematically carried out in order to effect a cure, or even an arrestment and quiescent state of the disease. Cod-liver oil, tonics, and alteratives should be prescribed. Thyroid extract may effect an improvement, or even a cure. Koch's new tuberculin may be tried. Locally, too much interference is to be deprecated. Sedative lotions are beneficial, and if there is exuberant granulation, caustics may be cautiously resorted to.

Syphilis of the nose is but rarely seen as a primary affection. Secondary syphilis is hardly ever found, except in the form of excoriations about the orifice. Tertiary manifestations are not uncommon, and take the form of infiltration, with ulceration of the mucous and submucous tissues, which may lead to necrosis of bone and deformity. There is sometimes great destruction of all the tissues, with much fœtid discharge. Similar effects may be met with in the inherited disease. Tertiary syphilis of the nose is usually met with in cases which have received inadequate treatment in the early stages. Mercury and iodide of potassium must be given at first, and latterly full doses of the iodide. Locally, cleansing antiseptic lotions are to be employed. Operative measures, as the removal of dead bone, must be cautiously resorted to, if at all.

Rhinoscleroma is an extremely rare, chronic, infiltrating granuloma. The nodules occur as hard, red, smooth plaques, sharply local, and with a tendency to slow advancement. Beginning beneath the nostrils, they may creep gradually up, accompanied by brawny induration of the pertaining skin, causing thickening of the nasal membrane and obstruction of the passages. There is no ulceration. The prognosis is unfavourable, and all that treatment can effect is the relief of the nasal obstruction as far as possible. The disease may even spread to the pharynx and larynx, and cause urgent dyspnœa.

EPISTAXIS.

Bleeding from the nose is found in connection with a great many diverse conditions. It occurs as a symptom in many of the diseases of the nose: in rhinitis, in specific ulcerations, and in cases of tumours and foreign bodies. It may occur in connection with various disorders, such as heart or lung disease, liver disease, purpura, chlorosis, and especially hæmophilia. Typhoid, measles, typhus, and other infective diseases are often associated with some epistaxis, especially in their early stages; in diphtheria it is common, and invariably accompanies the nasal form of disease. Sexual influences also induce it, and it appears to be undoubtedly vicarious of menstruation in many cases. Traumatism is a well-known cause. Males are the chief sufferers. Some young people are subject to violent recurring attacks, which give rise to much alarm. These are often of a compensatory nature, and hence not to be regarded as necessarily injurious, or otherwise of bad omen.

A person suffering from epistaxis should be placed in a semi-recumbent position on a couch or chair, the neck bared, and the feet placed in hot water. The head should be held slightly forward so as to enable the blood to get out by the nostrils, and prevent it flowing back into the mouth, the arms should be raised, and cold water or ice applied to the nape of the neck and over the nose. A plug of cotton-wool should then be pressed into the nostril impregnated with solution of alum, or diluted perchloride of iron. In most cases these measures will be effectual. If the hæmorrhage continues, plugging must be resorted to. If plugging anteriorly fails, posterior plugging must be effected. The plugs may be saturated with dilute perchloride of iron or hamamelis, or iodoform gauze may be used. Irrigation with hot water (120° F.) should be tried if necessary. Constitutional conditions should receive appropriate treatment.

NASAL POLYPI.

These are soft, pedunculated, fibromatous, or myxomatous tumours, growing usually from the middle turbinals. They apparently frequently coexist with suppurative catarrh of the accessory nasal cavities. Polypi rarely occur singly. They grow in numbers, and are frequently bilateral. The mucous membrane assumes a chronic myxomatous condition, so that as a rule the polypoid growths continually recur. Polypi must in all cases be removed either by the galvano-caustic or the cold snare. The operation requires to be carried out very thoroughly.

Other non-malignant growths are sometimes met with, such as papillomata, angeiomata, enchondromata, and osteomata. They all require operative treatment.

Malignant growths of the nasal cavities are found occasionally, in the form of epitheliomata and sarcomata. If recent, some few cases may be benefited by operation, but in the vast majority palliative treatment is alone called for.

FOREIGN BODIES.

Rhinoliths or nasal calculi are formed by the precipitation of the calcium and magnesium salts in the nasal secretion, either round a foreign body or piece of mucus, but occasionally no nucleus is discoverable. There are symptoms of obstruction with muco-purulent discharge, and often associated symptoms referable to the ear, eye, or head. Ulceration and perforation of the septum or face may even result. For rhinoliths and foreign bodies generally, the only remedy is extraction by one or other of the operative procedures in vogue.

DISEASES OF THE NASAL SEPTUM.

The principal affections of the septum are abscess, ulceration, deviations, and spurs. Abscess is usually due to traumatism, but it may also arise in connection with syphilitic or other infective processes. The swelling over the septum is very marked, and the external tissues are inflamed and swollen, often presenting an erysipelatous character and producing severe constitutional symptoms. Palliative treatment should at first be employed, and if signs of suppuration appear, a free incision should be made.

Ulceration of the septum is met with chiefly as a result of some infective process, *e.g.* syphilis, tubercle, lupus, etc., but may also apparently occur as an idiopathic affection. It not infrequently causes perforation. Syphilitic perforation is usually attended with much pain and discharge, and unless quickly arrested involves a considerable area. The treatment of ulceration is directed in the first instance to cleansing of the passages, then iodoform or the ung. hydr. nit. may be applied over the ulcer. Failing this, the galvano-cautery should be used, or the acid nitrate of mercury carefully applied.

Deviations of the septum are of frequent occurrence in adults, and own a variety of causes, including traumatism, defective development, and obstruction of one nostril. They are often accompanied by bony ridges and spurs. The ridges often occur along one or other of the sutural lines of the vomer. The projections known as spurs are either cartilaginous or osseous, and due to productive inflammation. The symptoms of deviations and spurs are those of nasal stenosis, namely, mouth breathing, nasal and

Eustachian catarrh, with more or less deafness, tinnitus, and frontal headache. Treatment is as a rule satisfactory. Slight cases may be remedied by the measures appropriate to a thickened condition of the mucosa, and by mechanical interference, with the object of straightening the septum. Forcible pressure by means of bougies, tampons, or specially constructed forceps often succeed. Spurs and crests have to be removed by operation.

DISEASES OF THE SINUSES.

The frontal, ethmoidal, and sphenoidal sinuses are rarely affected, though suppuration is sometimes found as the result of a catarrhal process, which is usually associated with disease in a neighbouring part, or with obstruction of the apertures. Suppuration of the maxillary sinus, or antrum, is on the other hand not uncommon, and may be considered more in detail.

Etiology and morbid anatomy.—Inflammation with suppuration of the antrum is usually the result of decayed teeth. A periostitis is originated and maintained around the roots of the affected tooth or teeth, which extends to the antrum, often suppurating as it proceeds. In some other cases the disease extends from the nasal fossæ. The mucous membrane of the antrum is inflamed, and at first secretes a more or less serous fluid. The fluid so formed may in part escape by the ostium. The membrane gets progressively thickened and infiltrated, and the secretion becomes purulent. It is invaded by pus organisms, and is converted into a "pyogenic membrane."

Symptoms.—The involvement of the antrum, if acute, is shown by sharp shooting pains, with redness and swelling of the cheek over the affected portion. In the more ordinary chronic cases these symptoms are absent, or much modified. Constitutional symptoms, in the form of some pyrexia, with malaise, etc., supervene. Slight rigors may indicate the onset of suppuration. The pus so formed commonly overflows into the nasal cavity through the natural aperture, and causes a foul-smelling discharge from the nostrils. It sometimes also passes backwards into the naso-pharynx. If the ostium is blocked, however, the pus distends the antrum, and gives rise to much swelling and pain locally.

Diagnosis.—Cases of empyema of the antrum, in which the pus escapes freely into the nasal cavity, may present some difficulty in diagnosis. If, on examination, pus is seen to escape from the middle meatus, it may originate from either of the sinuses, but the history and associated symptoms will be helpful. In chronic rhinitis no such discharge will be found. Trans-illumination of the antrum by means of electric light will often clear up the diagnosis. The operation of perforation of the antrum from the nose is a safe and effectual test in suitable cases.

Treatment.—Empyema of the antrum is nearly always amenable to cure by operation. The usual method is to open into the cavity from the alveolus: a tooth, or part of a tooth, if decayed, being first of all removed. After draining out the pus, thorough irrigation is effected by means of a suitable syringe (Higginson's). A drainage tube is afterwards inserted, and the cavity washed out from time to time by an antiseptic solution. In obstinate cases the mucous membranes of the cavity may require curetting. Other methods of opening into the antrum have also been advocated.

A. G. AULD.

AFFECTIONS OF THE NASO-PHARYNX.

Catarrh of the naso-pharynx is evidenced by a sense of dryness and soreness in the throat, and by a troublesome hawking of tenacious mucus. Examination by the rhinoscope may reveal swelling of the pharyngeal bursa or tonsil. Frequently, however, the affection is associated with nasal catarrh, or other nasal disorder. The treatment to be adopted, in the first instance, is the application of astringent and antiseptic solutions, as in *chronic rhinitis*, by means of the spray or brush. Applications containing iodine are useful. Chronic and intractable cases require cauterisation, or even excision, of the pharyngeal tonsil.

ADENOID VEGETATIONS.

An hypertrophy of the lymphoid tissue of the naso-pharynx, forming sessile or pedunculated growths.

Etiology and morbid anatomy.—The period of childhood and youth is specially that of adenoid growths. A family tendency, especially where tubercle exists, is often observed. Adults are sometimes affected. Chronic naso-pharyngeal catarrh precedes and excites the condition. The vegetations consist of a somewhat scanty reticulated connective tissue, enclosing lymph corpuscles. Ciliated columnar epithelium clothes their surfaces.

Symptoms.—The growth may be limited to the pharyngeal tonsil, when the symptoms are not so severe, but frequently the lateral aspects of the pharynx are invaded, giving rise to characteristic severe symptoms. Inspection then reveals a large lobulated mass, or several masses, extending from side to side of the pharyngeal vault. In a large percentage of the cases, hypertrophy of the faucial tonsils coexists. The face of the patient has a vacant expression, the mouth is kept open, the nostrils are pinched, and the chest becomes deformed. The voice is "dead" and nasal in quality, and articulation is defective. Persistent and often bloody nasal catarrh, with mouth breathing of a rough character, nocturnal snoring, "terrors," and auditory troubles, are characteristic. There is often persistent deafness.

Naso-pharyngeal obstruction seriously affects growth and the conformation of the chest. The lower lateral aspects of the chest wall are retracted during inspiration. This leads to protrusion of the sternum, forming the characteristic pigeon breast. In other cases, there is marked depression of the lower part of the sternum, giving the chest a funnel-shaped appearance; in yet other cases, asthma develops, and produces the barrel chest of emphysema. In the subjects of this affection the intellectual functions are much impaired. There is inability to concentrate the ideas, inattention, forgetfulness, stupidity, and disinclination for work.

Diagnosis and treatment.—This is rendered easy by the distinctive symptoms and by the detection of the growths on rhinoscopic examination, or on palpation. Under proper treatment the prognosis is distinctly good. This consists in removal of the growths by operation. Constitutional remedies are generally also indicated, in the form of iodide of iron, cod-liver oil, and change of air to the seaside.

Polypi, adenomata, cysts, and *malignant growths* are occasionally met with in the naso-pharynx.

A. G. AULD.

DISEASES OF THE LARYNX.

The larynx is examined by means of a reflector and laryngeal mirror. The observer seats himself in front of the patient, and attaches the reflector to his forehead in such a manner as to bring the pupil of the eye (generally the right) exactly opposite the opening in its centre. Sometimes, however, the reflecting mirror is placed over the middle of the forehead, and the opening in its centre discarded for the time being.

A strong light is placed behind and on a level with the patient's ear (generally the left) about four inches outwards. The head of the patient is then thrown backwards, the mouth widely opened, and the protruded tongue grasped in a small towel thrown over the operator's fingers, and gently pulled forwards and downwards. Then the laryngeal mirror is warmed over the flame, and its temperature tested by the observer applying it to his cheek. Thus warmed, it is held penwise, and introduced, without touching any part, right into the pharynx, and gently pressed upwards and backwards against the uvula and soft palate. The light from the reflector is centred on to the pharynx, and the patient directed to say "ah." Thereupon the larynx is raised and its various structures are brought into view, forming an inverted image in the mirror; that is to say, the epiglottis is seen in the upper part of the mirror, and the posterior portion of the larynx in its lower part, appearing respectively posterior and anterior. On phonation, the white or pink-white vocal cords are seen close together, while on inspiration they go apart, and the glottis is well open. Immediately outside the vocal cords, the red ventricular bands are seen. By manipulation of the mirror, the general state of the tissues, and the free motility or otherwise of the vocal cords, are thus to be clearly ascertained. In irritable subjects, a 20 per cent. solution of cocaine may be previously applied to the throat.

LARYNGITIS.

Acute Laryngitis.

A catarrhal inflammation of the mucous membrane of the larynx, often associated in children with local nervous disturbance.

Etiology and morbid anatomy.—Exposure to cold, inhalation of irritant substances, and overuse of the voice are the common exciting causes, but constitutional conditions usually lay the foundation for the disease, such as debility, rickets, or gout. It may occur as an extension from the naso-pharynx. It is also found as an occasional complication of one or other of the exanthemata. The membrane is red from injection, slightly swollen, and a scanty viscid mucus is secreted.

Symptoms.—Hoarseness, more or less aphonia, and tickling cough are the characteristics of mild attacks in adults. In more severe cases there is some febrile disturbance, pain and sense of constriction in the larynx, and embarrassed breathing. There is also much huskiness of voice and difficult hawking of sticky mucus. In young children the affection is of a peculiar type, inasmuch as, with but slight previous symptoms of catarrh, sudden suffocative attacks supervene, and hence the terms spasmodic laryngitis and false croup. The child is observed to have a hard croupy cough and somewhat impeded breathing during the day or evening. Then, some time after falling asleep at night, the little one awakens in a suffocative fit, evidenced by hard metallic cough, stridulous inspiration, and the signs of severe dyspnœa. The fit lasts usually but a few minutes, and the child goes to sleep again, but the attack may recur either that night or the following night or two. The cough and symptoms of catarrh persist throughout and for some time after the spasmodic attacks have ceased altogether. But spasm is not always the cause or the sole cause of the attack; swelling of the mucosa, paresis of the intrinsic muscles of the larynx, and accumulation of secretion are also causative agents. Laryngoscopic examination reveals redness and swelling of the mucous membrane, with impaired motility of the vocal cords, and more or less mucous deposit.

Diagnosis.—In adults this is simple enough, but in children we have to distinguish the affection from membranous laryngitis or true croup on the one hand, and from laryngismus stridulus on the other. True croup is, however, stable in its symptoms and regular in its progress, and the exudation is membranous in character, though it sometimes happens that intermediate cases occur which cause perplexity. Laryngismus stridulus, also often termed "false croup," is a purely nervous spasm, unaccompanied by previous cough and signs of catarrh.

The prognosis is as a rule good, provided watchful attention and sound treatment be given.

Treatment.—The patient must be kept in a warmed room, and his immediate atmosphere kept moistened by the steam-kettle. Bland liquid diet should alone be given. The bowels must be kept freely open, and warm poultices applied over the larynx. Some prefer the ice-bag. Sprays containing benzoin, or 5 per cent. solution of menthol in paroleine, may be applied to the throat. Lozenges of cocaine and rhatany relieve the irritation. When, in children, suffocative attacks occur, a warm bath is to be employed, and hot applications, as a sponge wrung out of hot water, applied over the throat. Chloroform inhalation may prove serviceable, and an emetic of ipecacuanha wine may be given with benefit. Should life be endangered, intubation or tracheotomy must be resorted to.

MEMBRANOUS LARYNGITIS—CROUP.

A severe inflammation of the mucous membrane of the larynx, characterised by the formation of fibrinous membrane, and also by coagulation necrosis of the mucous epithelium.

The disease has been much misunderstood, and confusion has arisen. But the progress of bacteriology has placed it outside the simple inflammations, and shown it to be diphtheritic in character, or else true diphtheria.

Etiology.—Membranous laryngitis is in a large number of cases nothing more or less than diphtheria. The acute formation of membrane, with coagulation necrosis, is always due to a poison of a corrosive character, and, when not diphtheritic in origin, is associated with the presence of virulent streptococci, or caused by powerful chemical irritants. Hence membranous laryngitis may, so far as we know at present, be the outcome of several severe poisons, the most common of which is that of diphtheria. It is pre-eminently a disease of childhood, and is accompanied as a rule by high fever, by croupy cough, huskiness of voice, and symptoms of dyspnœa. Cyanosis is marked in most cases, and sudden choking fits arise, with much restlessness and general distress.

Diagnosis and prognosis.—Its grave persistent symptoms distinguish it from simple acute laryngitis, though, if the latter is severe, it may be difficult or impossible to make a diagnosis at first. Many cases of severe laryngitis in children have been confounded with membranous croup. Laryngoscopic examination (if possible to be made) may clear up the case. Grey deposit should be looked for on the fauces, and if found, the disease should be regarded as diphtheria. This will be confirmed if the diphtheria bacillus be also found. The urine should be examined for albumin. The prognosis is grave, and with the youth of the child the gravity increases. An early treatment with the diphtheria antitoxine improves the prognosis.

Treatment.—The general directions given for simple laryngitis are more or less applicable to the membranous variety. The throat may be sprayed with warm corrosive sublimate solution (1–2000), and, if old enough, the patient may be able to inhale it. The steam atmosphere is generally employed, but its effects may be weakening, and it should be cautiously used. Judiciously employed, it may give great relief. The highest importance is to be attached to rigorous supporting measures—beef-tea, milk, and stimulants (whisky and water, champagne). Many cases will require intubation or tracheotomy. It remains to be said that, *as soon as the disease is recognised*, the diphtheria antitoxic serum should be given, and its effects watched, or, in certain circumstances, the antistreptococcic serum may be tried.

CHRONIC LARYNGITIS.

A chronic catarrhal condition of the laryngeal mucous membrane.

Etiology.—Persistent use and straining of the voice, as in public speaking, the inhalation of irritant vapours, as tobacco smoke and atmospheric dust, and repeated acute congestion, are the main causes.

Morbid anatomy and symptoms.—The entire mucous membrane is swollen, from injection and exudation into its substance, and there is considerable formation of adhesive mucus. The vocal cords are especially congested, and sometimes there are localised thickenings, involving the cords or ventricular bands. Hoarseness and more or less aphonia are characteristic. Discomfort is felt in the throat, and a desire to clear it frequently, when a little sticky mucus is often expectorated. In a variety of the affection known as *laryngitis sicca*, the exudation is very scanty, and inclined to form crusts; while in another form, *laryngorrhœa*, much secretion is expectorated, especially on using the voice.

Diagnosis.—In addition to the symptoms complained of, laryngoscopic examination renders this easy. The nasal passages should always be examined, as obstruction therein may coexist and may even have induced the laryngeal condition. With proper care and treatment the prognosis is favourable, but otherwise the affection may be very intractable.

Treatment.—Rest of the voice is a *sine quâ non*. Tobacco must be prohibited, or used in great moderation. The same applies to strong or spiced foods and drinks. General treatment is to be adopted in all cases where possible, particularly change of air and bracing measures. Preferably the mountain air, in a mild, equable climate, is to be selected, and this may be profitably combined with a course of waters, such as those of Ems, Aix-les-Bains, or Mont-Dore. Locally, the patient may be taught to use the spray, inhaling at first, twice or thrice daily, solutions of pine oil, menthol, eucalyptus oil, or chloride of ammonium, and afterwards alum or chloride of zinc (2 or 3 grs. to the ounce). Insufflations of bismuth may do much good. If the case is very obstinate, the larynx should be painted by the brush with the chloride of zinc solution or nitrate of silver solution (10 to 30 grs. to the ounce), lactic acid (30 to 50 per cent.), or such like specially recommended preparations.

ŒDEMA OF THE LARYNX.

Etiology.—Œdema of the larynx may complicate general œdema, as in kidney and other diseases, or it may apparently arise very acutely, either as a neurotic manifestation, or as a complication of some acute general disease, *e.g.* scarlet fever, typhoid. More frequently it is associated with local or neighbouring disease, as in the pharynx. It is then often found that the disease in question is of a septic or erysipelatous character. But laryngeal catarrh, perichondritis, and ulcerative conditions of the larynx are often productive of more or less localised œdema. Sometimes the œdema goes on to abscess. It must always be regarded as a serious condition.

Symptoms.—Rapidly increasing dyspnœa, stridor, and huskiness of voice, proceeding to aphonia, are diagnostic. There is great swelling of the epiglottis, which may often be felt or even seen. The deep œdema specially involves the ary-epiglottidean folds.

Treatment.—Leeches may be applied over the larynx, followed by ice-bags. The atmosphere should be moistened by steam, and ice given to suck, these measures being not necessarily antagonistic. Hypodermic injections of pilocarpine ($\frac{1}{6}$ gr.) may be given. Scarification should be resorted to if there is any urgency of the symptoms, but intubation or tracheotomy may be required. Internal remedies must be administered, if there is evidence of constitutional infection of any kind. Perchloride of iron is very useful for erysipelatous cases, and the antistreptococcic serum might be tried.

INFECTIVE GRANULOMATA.

Tuberculosis of the larynx, or laryngeal phthisis, is sometimes primary, but as a rule it is secondary to pulmonary tuberculosis. In the earlier stages the ary-epiglottidean and interarytenoid folds are usually swollen in some of their portions, in an irregular fashion, from infiltrated tubercle: or actual tumour-like outgrowths occur. Yellow spots or patches may often be seen in the thickened tissue. The vocal cords are often similarly affected and split up into ridges, or, in other cases, it may be the epiglottis which shows the characteristic swelling. Ulceration supervenes, giving rise to much purulent secretion, with deep destruction of tissue, and imparting a ragged appearance to the cords, and often also to the ventricular bands. The cartilages may latterly become implicated in the ulcerative process. The diagnosis is usually easy, from the local appearances, associated symptoms, and the presence of tubercle bacilli in the expectoration, though syphilitic disease might mislead in some cases. But in syphilis congestion is more marked. In tuberculosis there is often a striking anæmia of the tissue, especially in the early stage. Syphilitic ulcers, moreover, are larger, spread rapidly, and are prone to attack the upper aspect of the epiglottis. The treatment must of course be principally constitutional. While the disease can rarely be prevented from proceeding to a fatal termination, much relief can be given by local applications. Of these, lactic acid is best. Cocaine must be thoroughly applied first, then a 20 per cent. solution of lactic acid is to be well rubbed over the tissues, preferably by a cotton-wool pledget, but a brush may be used if desired. Day by day this is to be done, gradually using a solution of greater strength, until 80 or 90 per cent., or even full saturation, is reached. After two or three weeks, the ulcers may be healed and cicatrisation brought about. A long-continued course of pure creasote, beginning with 3 minims and leading up to 15 minims (in capsules) three times a day, is recommended by Semon.

Lupus of the larynx is distinct from what is known as laryngeal phthisis, notwithstanding that the two affections are supposed to be due to the same micro-organism. If so, there are modifying conditions not yet understood. Lupus of the larynx is not a common disease. It is almost invariably associated with lupus of the skin, but a few cases of primary infection have been recorded. It appears as nodule-bearing thickenings of the epiglottis (most commonly), but any part may be attacked. The nodules are small and somewhat thickly set, and they ulcerate slowly. Pain is slight or absent. The ulcers have this peculiarity, that they cicatrise, and hence furnish a valuable aid in diagnosis, though the presence of the disease in the skin is usually enough. Constitutional treatment, as for lupus generally, is to be mainly relied upon. Thyroid extract may be tried. Koch's new tuberculin may prove remedial. The lactic acid treatment, as given for tuberculosis, should also be adopted. The electric cautery and other caustics have been employed in some cases.

In the commencement of the secondary stage of syphilis there is usually some congestion of the larynx, causing a roughness or hoarseness of the voice, which to a practised ear is somewhat characteristic. Occasionally mucous patches or condylomata occur. These conditions quickly pass away under mercurial treatment, and no local measures are required as a rule. Tertiary syphilis is found usually in the form of ulceration, which commonly attacks the epiglottis; this is preceded by swellings, due to infiltration or gummata. At first the swellings are red and inflamed, then a yellow spot appears upon them, which extends, leading to rapid and deep ulceration, so as often to involve the cartilages. The danger does not end here; for, after healing of the ulcer, cicatricial tissue forms, sometimes as bands extending across the cords, and forming other adhesions, all of a dangerous character. The prognosis is so far favourable, as thorough constitutional treatment may cure the ulceration, especially full doses of the iodide of potassium, but the deep ulcerations and stenoses are serious. The latter may require gradual dilatation or tracheotomy.

PERICHONDRITIS

As already pointed out, the cartilages of the larynx may be involved in tuberculous and syphilitic ulcerations. They are also commonly attacked in malignant disease. Primary perichondritis is also alleged to occur, though complete proof is perhaps awanting. The necrosis of the cartilages (which is the outcome of inflammation) is sufficiently shown sometimes by the expectoration of portions; or laryngoscopic examination and the introduction of a probe may reveal the exfoliated cartilage. The arytenoids are most frequently attacked. When due to syphilitic disease, the prognosis is somewhat favourable; but under other conditions it is grave, and tracheotomy may be required.

NEW GROWTHS

Papillomata and fibromata are much the commonest. The former are small, warty growths, generally aggregated in clusters. Fibromata, on the other hand, are solitary, and sometimes pedunculated. These growths occur usually on the vocal cords or ventricular bands, but they may grow on other parts. Other simple tumours, such as cysts or lipomata, are very rare.

The prognosis in the case of these tumours is good; papillomata, however, tend to recur. They are generally accompanied by irritation, cough, and some alteration in the voice. When large, they give rise to dyspnœa. The treatment in all cases is that of removal by one or other of the operations in use.

Cancer and sarcoma are both found in the larynx. Epithelioma of the larynx is a common affection, and is usually primary. It begins as a small warty growth, generally on one or other vocal cord or ventricular band, but any part may be attacked. The wart has a broad base, and there is some redness and swelling of the tissues surrounding it. In the case of the vocal cords, that which is attacked is not only congested, but—a point of great importance from a diagnostic point of view—its mobility is impaired. It remains more or less fixed by the deep extensions of the growth. Epithelioma has also a tendency to extend backwards towards the arytenoid cartilages. The symptoms of the disease are hoarseness, aphonia, and often shooting pains in the direction of the ears, and, in advanced cases, glandular enlargement in the neck; but this may be contingent on the situation of the growth. It is unnecessary here to enlarge further on this subject, but it may be said that the diagnosis is often attended with great difficulty. Of course, in the advanced stages of ulcerative destruction, this is rendered easier, though syphilitic disease may in some cases prove a stumbling-block. The result of treatment will usually suffice to clear this up, however; but it has to be remembered that cancer may be grafted on the syphilitic disease. Again, some apparently innocent conditions may ultimately become cancerous. If the patient be elderly, and a history of aphonia and hoarseness lasting for months, or even for a year, be obtained, and if, on examination, a tumour or tumour-like infiltration be found, possibly of tough consistence and accompanied by circumscribed congestion, then, even without much alteration in the mobility of the cord (though this will in some degree depend on the situation of the growth), even without pain locally or constitutional signs, the case is to be regarded as in the very highest degree suspicious. If possible, a small piece of the growth should be removed by forceps for microscopic examination.

As regards treatment, the methods adopted are—endolaryngeal removal, partial resection, and complete excision of the larynx. As regards their justification or utility, that is another matter. Partial resection is justifiable in some cases. If the disease is not causing any respiratory trouble, it is perhaps best to leave it alone, employing palliative measures; when signs of obstruction arise, tracheotomy is to be performed. It must also be borne in mind that frequent meddlesome interference is wrong, and may hasten or even induce the malignant condition.

Sarcoma of the larynx is rare, but is more amenable to operative treatment than carcinoma.

NEUROSES OF THE LARYNX.

This is a wide and complex subject, and little more can be done here than to make mention of the principal conditions. Of these the most important is the spasmodic affection of the adductor muscles, known as *laryngismus stridulus*, which has already been incidentally referred to in connection with acute laryngitis. It is found in young children, most commonly before the third year, and is nearly always associated with some constitutional disorder, such as rickets. Teething and gastric disorder frequently induce the attacks, which come suddenly, and often without warning. The choking is generally of an agonising character, and death seems imminent; but milder cases occur. Various other convulsive seizures may accompany the laryngeal spasm, *e.g.* carpo-pedal contractions. The attacks are of variable duration, but death seldom occurs, the spasm suddenly relaxing just as the patient, bedewed with cold sweat, seems to be moribund. The affection appears to be cortical in origin. Treatment must be very prompt. The child should be placed in a warm bath, the clothing having been removed, especially any close-fitting garment. The windows should be thrown open, and cold water dashed over the face and chest. Smelling-salts may be held to the nostrils, and, if at hand, ether, amyl nitrite, or vapour of chloroform cautiously administered. Should these measures prove futile, artificial respiration should be adopted, and, as a final resort, tracheotomy should be quickly performed. As a general rule, however, the ordinary simple measures will prove sufficient. In those subject to the attacks, the general health should be perseveringly watched, as regards diet, digestion, etc., the bowels being kept open, and appropriate constitutional remedies given. Laryngeal spasms are also met with sometimes in adults, under the conditions of hysteria, chorea, epilepsy, locomotor ataxy (laryngeal crises), and some other disorders. Certain primary spasmodic neuroses are also found, namely, *mogiphonia*, the analogue of writer's cramp; laryngeal vertigo, a sudden spasm of the larynx during a coughing fit, accompanied by vertigo and transient syncope; and the so-called barking cough of puberty. The treatment of these various conditions, often tedious, can only be satisfactorily indicated after thorough examination of each individual case, and can hardly be profitably entered into here.

Paralytic affections of the larynx form a numerous group, and their pathology is very intricate. Much has been recently done, however, in this department of neurology, for which the reader is referred to special works. The most important forms are the paralyses of the adductor and abductor muscles. The adductors, or the muscles which close the glottis, are subject to paralysis in hysterical females, constituting the affection known as hysterical aphonia. Rarely does the affection otherwise arise. The treatment is that for hysteria generally, with the application of electricity locally, the faradisation being used both outside and within the larynx, if need be. Paralysis of the abductors, or the muscles which open the glottis, is either bilateral or unilateral. The bilateral variety is generally due to pressure by tumours upon the pneumogastric or recurrent laryngeal nerves, but central lesions—degeneration, tumours, etc.—are also causative, and likewise specific toxines, as those of diphtheria and scarlet fever, which cause neuritis. It is attended by much inspiratory dyspnœa, and tracheotomy may be required. Unilateral paralysis owns a variety of causes, both central and peripheral. Of the latter, pressure on the left recurrent nerve by an intrathoracic aneurysm, causing left-sided paralysis, is much the most common. The treatment of these forms of paralysis must be directed to the cause. Iodide of potassium may often be useful. Local treatment is not of much avail.

A. G. AULD.

DISEASES OF THE TRACHEA AND BRONCHI.

ACUTE BRONCHITIS.

TRACHEO-BRONCHITIS.

An acute catarrhal inflammation of the mucous and sometimes also of the submucous structures of the trachea and bronchial tubes.

In the early part of the century the term bronchitis was applied to catarrh of the bronchi, but before Laennec's time the affection was confounded with pulmonary and pleuritic conditions. It was termed "bastard peripneumony" by Sydenham, to distinguish it from true peripneumony, or what is now termed pleurisy. It was supposed due to a collection of humours in the blood, which ultimately gathered in the lungs, inflaming them, and seeking escape by the bronchial passages.

Etiology.—The young and the old are most liable. The principal general exciting cause of the disease is undoubtedly a cold, variable, and moist climate. Thus Great Britain (and particularly Scotland) affords an ideal field for its existence, and it is probable that bronchial affections form the commonest class of disease in this country. Warm, equable, and dry climates, on the other hand, are unfavourable to its appearance. The north and east winds are particularly harmful, though it is just possible that the east wind gets more abuse than is its due. Sudden changes of temperature, either in the weather, or from artificial causes, are particularly dangerous. Thus, going from heated apartments into the cold night air, often without sufficient wrapping, is a fertile cause of acute bronchitis; and if the hot air is also impure, either from overcrowding, gas, or any other cause, the danger is enhanced. In like manner, the transition from cold to hot air entails irritation of the bronchi, and may lay the foundation of an acute attack. Sitting or standing in cold draughts of air is probably, however, the most common of all exciting causes. But though this is so, it is a remarkable fact that strong and healthy persons are hardly ever attacked, notwithstanding their utter disregard of the usual exciting causes. It consequently has for long been apparent to physicians that a constitutional defect must be reckoned with in most examples of the disease. There may be an hereditary tendency to catarrh of mucous membrane. The scrofulous constitution also entails a vulnerability of the various mucous surfaces, as seen in the liability of scrofulous subjects to catarrh of the conjunctivæ, throat, etc. Of other underlying constitutional states, gout, rheumatism, Bright's disease, and rickets are those mostly encountered. Senility of the tissues, especially in those of a comparatively young age, affords a potent inducement for the disease. This may be revealed by atheroma or calcareous degeneration of the arteries.

Acute bronchitis is also, in quite a large percentage of cases, grafted on the chronic variety, or some other pulmonary affection, especially emphysema and asthma. In addition to these factors, we have, as predisposing causes, many conditions of acquired disease or defect. General debility, however induced, may be instanced in this connection. Heart disease, and especially mitral disease, causes a congested condition of the bronchial mucosa, which may precipitate acute bronchitis. Acute bronchitis may complicate many of the exanthemata, particularly measles and whooping-cough. It is sometimes found in the initial stage of typhoid, and nearly always in that of typhus.

Morbid anatomy and pathology.—In ordinary tracheo-bronchitis the mucosa is highly congested and considerably swollen, and as a result serous exudation shortly ensues. The nerve fibres, and especially the ganglia at the bifurcation of the trachea, are irritated by the pressure, and so pain and cough are produced. Then the epithelium lining the tubes becomes swollen, and many of its cells are transformed into goblet cells. Similar changes are found in the mucous glands, and from these two sources large quantities of mucus are generated. Leucocytes gradually accumulate in the mucosa, and many of them are extruded through the minute canals or foramina which traverse the basement membrane. The next step in the process is the division of many of the columnar surface cells. Their nuclei show karyokinesis, and as a result the cells divide into two, each part having a separate nucleus, though occasionally three segments are produced. In addition to this, the layer of round cells next the basement membrane proliferates abundantly. In many cases of acute bronchitis the changes end here, and the cells undergo fatty regression. But severe forms are further characterised by division of the fixed connective tissue corpuscles in the bronchial wall, including the adventitia.

The pathology of the disease is not so clear. It is certain that many cases of acutely inflamed bronchi are found, especially in connection with specific diseases, which are characterised by an abundance of micro-organisms present in the mucosa. So far, this is but natural in the case of a structure so exposed to the contact of the air. But there can be little doubt that acute bronchitis, in its typical or classical form, has many characters of a specific disease. Its invasion, widespread extension, assembled symptoms, acme, and more or less critical decline, are suggestive. Whether amongst the micro-organisms which have been described in the mucosa a specific bacillus has ever been seen, may be possible, but nothing definite can be pronounced on the subject at present.

Symptoms.—In acute tracheo-bronchitis the temperature rises to about 100° or 101° F. in adults, but in children it is a degree or two higher. Respiration is more or less embarrassed, and there may be attacks of spasmodic dyspnœa. A sense of constriction and more or less pain are complained of in the chest. At first the cough is dry, hard, painful, and wearisome. It causes exhaustion in many cases, and general tremulousness from its concussive action on the nervous system. Sleep is also much disturbed. Expectoration, at first absent or scanty, gradually increases, and consists of a colourless, fluid, and frothy secretion, intermixed with strings of coagulated mucus, and sometimes streaky with blood. As the disease advances the expectoration assumes a greenish colour, is less frothy, and acquires considerable viscidity. It averages roughly from 6 to 14 oz. in the day, but there is great variation in this respect. After a few days (4 to 10) the disease will commonly subside, the cough becoming easy, and only excited to expel the purulent mucus which now forms in the air tubes.

But severer cases occur, and these are especially found in the subjects of a chronic weakness in the air tubes, especially stout elderly persons. The dyspnœa is great, the auxiliary muscles being requisitioned, and cyanosis becomes marked. The breathing, too, is often accompanied by a more or less audible gurgling, produced by the passage of the air through the secretion-laden tubes. The pulse at first is frequent and bounding, and the temperature considerably elevated. Delirium is often present, especially during the night. In such cases the finer bronchi become involved, and we also find the symptoms of œdema of the air tubes, especially if any old emphysema coexists, and there is some œdema of the lungs as well, more particularly over the bases. All this may be present without any special weakness of the heart, though of course, in cases of cardiac debility or disease, the symptoms will be intensified, when dropsy of the extremities may occur, and even the trunk may be implicated. The general inflation of the lungs, brought about by the bronchial obstruction, often termed acute emphysema, will tend to impede the circulation, embarrassing the right heart, and so predisposing to the condition of œdema. In fatal cases, cyanosis becomes extreme, and expectoration fails. The extremities become cold, and there may be profuse perspirations. Unconsciousness is early developed (always a bad sign), and, needless to say, persists till the end.

The physical signs of acute bronchitis are definite and easy of determination in uncomplicated cases, but if the disease be superadded to some other affection of the chest, some difficulty may be encountered, as in the case of acute bronchitis occurring in the subject of an old fibroid consolidation. In ordinary bronchitis the chest on inspection is expanded, and sinks in but little during expiration. Both thoracic and abdominal movements are exaggerated or abnormal. The chest is lifted on inspiration, the inferior, part of the sternum often being simultaneously depressed. But the lower parietes and sometimes also the upper anterior portions may sink in on inspiration, if much obstruction exists in the tubes. As a rule, however, the upper part of the chest bulges. Expiration is slow and laboured. Palpation reveals increase of the vocal fremitus, and rhonchal fremitus may be present. The heart is usually displaced downwards and to the right. The liver is slightly depressed also. Percussion is normal, or, in more severe cases, hyper-resonant, from the general inflation of the lungs. The bases, if œdematous, will, however, yield a higher pitched, *i.e.* duller, note. Auscultation discloses a noisy character of the breath sounds, and *expiration is markedly prolonged*. The characteristic adventitious sounds are caused by the swollen and consequently rigid condition of the air tubes, by the presence in them of masses of secretion, and sometimes also by local spasm of their muscles. They consist at first of sonorous râles or rhonchi in the larger tubes, and of sibilant or whistling râles in the medium-sized ones. As the disease progresses, and the mucus increases and softens, the sounds become moister, and are referred to generally as mucous râles; but all kinds coexist, and come and go with coughing. In the smaller tubes the râles are termed submucous and subcrepitant. Congestion and œdema at the bases produce fine liquid crepitant râles, which, when very numerous, will obscure the vesicular breath sound.

Diagnosis.—The sudden invasion, cough, adventitious sounds, and absence of dulness over any region of the lungs, render the diagnosis generally easy. Acute tuberculosis of the lungs may have somewhat similar symptoms and physical signs at first. The progress of the affection and associated symptoms will determine the presence of tuberculosis, but it must be remembered that, in acute tuberculosis, bronchitis is present as a symptom. Acute tuberculosis and acute bronchitis may coexist. In like manner bronchitis may be developed as a primary affection in those suffering from old chronic affections—chronic pleuritis, chronic pneumonia—and it may also coexist with acute pleurisy. The presence and site of the râles and character of the expectoration will usually suffice to indicate the presence of bronchitis.

Prognosis.—Acute bronchitis in children will be separately considered, as it is apt to assume the characters of a grave affection. In the commoner forms of the disease in adults, the prognosis is favourable, but in elderly persons the disease is always to be regarded with some anxiety, especially in cases of weak heart, while in old age it is very fatal, and forms a large percentage of the deaths in this country. The condition of the patient's tissues and the presence or absence of disease elsewhere should be carefully considered. Particularly, the arteries are to be examined for atheroma, and great pains bestowed on the condition of the heart, in order to form an estimate of its general nutrition and working power. Any defect of the latter adds gravity to the prognosis. Acute bronchitis, superadded to the chronic affection, is likewise to be anxiously regarded. It is also important to remember that those who have once had an attack of acute bronchitis remain more or less liable to a recurrence of the disease, and a chronic affection is not infrequently set up.

Treatment.—As soon as bronchitis makes its appearance, such measures should be adopted as will favour its elimination. An arrest of its progress can only be effected by inducing diaphoresis, and by the application of hot fomentations and poultices over the chest. Steam may also be inhaled. Recollecting that the local inflammation is strictly defensive, and the natural means of combating the poisonous agents which are deposited and generated *in situ*, it is clear that local and general stimulation will tend to the dispersal and elimination of the toxic agents. Should, however, the poison gain ground, the local inflammation in the bronchial tubes, necessary for its destruction and elimination, will advance. The diseased products will then be thrown off in great part into the tubes themselves, and expectorated, whilst a part will be disposed of by the cells of the tissues.

It is common to give such substances as ipecacuanha, spirits of chloroform, or Dover's powder, at this stage, and these give relief from the more distressing local sensations. Opium, except when the cough is dry and irritable, should, however, be avoided if possible, as it tends to check the normal progress of the secretion. This, as already mentioned, is beneficial, and should rather be hastened than retarded.

In the early stage, an abundance of warm liquid diet (especially milk), diaphoretics, and hot poultices, should be employed. The bowels should be kept freely open. The temperature of the apartment should be maintained about 65°, and kept moist by steam. These measures are to be continued till the crisis of the disease is past.

When the disease assumes a sthenic form, antimony is often given, but probably it is better to trust to purgatives, blue pill being most suitable. In asthenic cases, on the other hand, stimulants are imperative. Alcohol, carbonate of ammonium, and strychnine are the most useful agents.

The secretion may, in debilitated subjects, become over copious, from inordinate œdema, and then belladonna should be administered in full doses, and the bowels kept active. Again, in children, the expulsive efforts may be insufficient. In such cases, an emetic of ipecacuanha should be given, as opportunity indicates. In adults, the heart may be feeble, when alcohol, digitalis, strychnine, and nourishing foods are to be relied upon. As the disease declines, tonics, as quinine and iron, and abundant nourishment, are the measures to be adopted.

Complications and special aggravations of one or other of the symptoms peculiar to the disease, may be met with. Spasm of the tubes demands such remedies as nitroglycerin, spirits of chloroform, opium, belladonna, lobelia, or ether. Great dyspnœa may require to be met by inhalations of oxygen, but commonly emetics and stimulants will suffice. Should cyanosis become threatening, venesection must be resorted to.

As already mentioned, it will often be found that a diathesis, or *materies morbi*, is present, which is either surely provocative of the disease, or else materially contributes to its advance. Such is to be sought for, and if found, to be met by appropriate specifics, *i.e.* the salicylates in rheumatic cases, alkalies and colchicum in cases of uric acid poisoning, with the diet appropriate thereto.

CAPILLARY BRONCHITIS.

Inflammation of the small bronchi occurs in the great majority of cases in an acute form, but there is also a chronic form of the affection.

Acute capillary bronchitis is chiefly found at the extremes of life, but in childhood it is generally associated with pulmonary inflammation as well, whereas in old age it is as a rule independent of such well-marked changes in the lungs. The inflammation often extends from the larger tubes, but may be otherwise set up by various agencies, and especially by infectious fevers or whooping-cough in the case of children. A highly purulent secretion is formed, which may completely block many of the bronchioles, and lead to collapse of the dependent lobules of the lung.

Symptoms.—The pyrexia may reach 104° F., but is very variable, and the respirations are extremely frequent. Dyspnœa is great, and symptoms of cyanosis are soon set up. Though this is so, the cough may be but moderate or even slight, and expectoration is scanty and muco-purulent. Sometimes, however, severe paroxysms of coughing, attended by high cyanosis, supervene. Great general distress, cold perspirations, and nervous twitchings are characteristic. The face is terror-stricken and pallid, except for a dusky patchiness over the cheeks. The extremities are cold.

The physical examination, while of course general, should be carefully directed to the posterior aspect of the lungs. More especially in children, the first indication of impending danger is often the development of fine crepitations, extending downwards over the bases from the scapular border. Percussing carefully over the bases may reveal a certain area of dulness, and if this is so, it is certain that broncho-pneumonia has been established as well. In other cases no dulness may be detected, and then the lung is partially permeable, but deep-seated patches of consolidation even then usually exist. It is scarcely necessary to add that the lower part of the chest is retracted during inspiration, and not infrequently there is depression in the supraclavicular region as well. Auscultation over the affected portions reveals moist crepitations accompanying inspiration. Towards the end of the act, some vesicular breath sound of a roughened character will be heard, if the lung has not become much involved. But if such cannot be heard, the fine râles being alone audible, then it is certain that the pulmonary substance is blocked, and careful percussion will generally reveal dulness over the affected part.

Acute capillary bronchitis is a most serious condition. Recovery, always tedious, may be looked for in those who are not very near the extremes of life. But in infants and aged persons recovery is rare, and, in the case of infants, should broncho-pneumonia be developed, a fatal issue is the rule.

Treatment.—The treatment of the affection must be very energetic. The apartment should be kept moist by steam. The great desideratum is the maintenance of the strength of the patient. Concentrated nourishment and alcohol are chiefly to be relied upon, given at frequent intervals. Beef-tea (made from fresh beef), milk, cream, and white of egg are best, if the stomach will receive them, otherwise the food must be peptonised. The chest and back generally may be rubbed with a rubefacient, and enveloped in wadding. Treatment by drugs must be regulated entirely by the symptoms as they arise. If expectoration is so defective that suffocative signs appear, an emetic of ipecacuanha may give relief, and oxygen gas may be directed over the mouth and nose. In some cases carbonate of ammonium will be found serviceable. Opium should be avoided. Lately, in children, it has been recommended to push belladonna until its physiological effects are fully produced, and so prevent the blocking of the air tubes by secretion. This method is still on its trial. The general directions given for ordinary bronchitis are more or less applicable for the symptoms of the capillary variety.

CHRONIC BRONCHITIS.

This disease is a chronic catarrhal inflammation of the trachea and bronchial tubes.

Etiology.—The factors which influence the production of chronic bronchitis are practically identical with those already discussed in connection with the acute variety. Underlying diathetic conditions are present in the majority of cases, especially emphysema, gout, and scrofula. Recurrent acute attacks, heart disease, kidney disease, and all constitutional and local forms of weakness, render the bronchial mucosa susceptible. In a great number of cases the affection develops yearly at the beginning of winter, and practically ceases on the advent of summer. Those whose occupations entail the inhalation of an irritant atmosphere are very liable to be attacked. Chronic bronchitis is almost exclusively confined to middle and advanced life, but especially the latter.

Quite a large class of cases of chronic bronchitis are purely secondary to pulmonary diseases. All chronic affections of the lung are attended by more or less chronic bronchitis. Broncho-pneumonia, phthisis, cancer, foreign body pneumonia, fibroid pneumonia, and primary emphysema may be cited in this connection. It is also produced by disease of the mitral valve.

Chronic bronchitis is often caused by the poisons of the various infective fevers.

Morbid anatomy.—The lungs are always more or less emphysematous, depending on the duration of the disease. The mucous membrane of the trachea, larger, and middle-sized bronchi is, in the majority of cases, much swollen, deeply injected, and coated with muco-purulent secretion. All the component structures of the tubes are found, in the earlier stages, to be hypertrophied. Often ridge-like longitudinal bands are seen in the trachea, composed of hypertrophied connective and elastic tissue, and the writer has seen remarkably developed nerves in the interior of the bands. The epithelial structures are at first hypertrophied, and exceptional cases occur wherein this epithelial hypertrophy is excessive, and amounts to a distinct abnormality. After the disease has lasted for many years, the tubes atrophy, and the epithelial structures are present merely in an embryonic form.

Symptoms.—It is a task of no little difficulty to give anything like a truthful representation of the symptoms of chronic bronchitis, for we find, in point of fact, that the affection occurs in most diverse forms, whose relationship to each other has not as yet been accurately settled. Laennec was the first writer who attempted a classification. Roughly speaking, he divided the disease into the dry, moist, and fœtid varieties. This classification is by many retained to the present day. Others again (*e.g.* Greenhow) have classified the various forms according to the diathetic or other tendencies which underlay them—as gouty bronchitis, rheumatic bronchitis, scrofulous or phthisical bronchitis, the bronchitis associated with skin diseases, kidney diseases, etc. Bronchitis outside this category was referred to as simple or uncomplicated.

Now it is evident that a chronic inflammation of the bronchi, as in the case of other chronic inflammations, is produced by the local action of an irritant, and whether that irritant be one or many, it does not matter much in so far as the symptoms are concerned, for these always are, and must always be, those of the defensive inflammatory process. But we certainly find a great variation in the degree in which one or other of the inflammatory manifestations are projected. Thus we sometimes find the quantity of serum or of mucus to be inordinately great, indicating much weakness in the vascular or mucous tissues, while in other cases the discharge is scanty, but there may be much local irritation and proliferation of connective tissue.

Whatever be the dominant feature shown by the inflammation, it will depend on the constitutional condition of the patient, together with the nature of the local irritant. And the symptoms themselves are to be truly regarded as the result of a process of defence and repair, defective often certainly, unless art steps in and gives a helping hand. The clear duty, therefore, of the physician is to endeavour on the one hand to discover the nature, or probable nature, of the irritant, and, on the other, to place the patient in the most favourable condition by the enforcement of both general and local measures.

In a great many cases of chronic bronchitis, the disease is very much limited to the trachea and larger bronchi. In such there is little if any constitutional disturbance. The cough is worrying at times, and there may be considerable uneasiness in the chest. Expectoration is scanty or moderate in amount, often largely confined to the morning, just after getting out of bed. Occasional exacerbations occur, especially in winter, and while the disease may last in this way for many years, it tends to assume a more severe aspect, unless special measures are adopted at an early stage. In this mild form of bronchitis the physical signs consist chiefly in the presence of rhonchi in the large tubes. Transient mucous râles may occasionally be heard over a limited area, but the smaller tubes are usually healthy. Emphysema is absent, or slightly developed.

On the other hand, there are cases in which the morbid process may be chiefly present in the smaller tubes, often more or less limited to certain districts of lung, when the "dry" variety of bronchitis is present. Herein there is much paroxysmal distressing cough, with scanty, viscid, pearly-looking sputa. Emphysema is developed, and mucous râles, sibili, and rhonchi are heard on auscultation.

But when the affection becomes established, in its more typically severe form, the subjective symptoms, both general and local, form a distressful incubus. There are persistive, though fitful cough and breathlessness, wearying restlessness by day and night, digestive disturbances, and a frequent sense of exhaustion. Malaeration of the blood is generally present in some degree. Inspection of the chest reveals more or less of the barrel shape which is pathognomonic of emphysema. Palpation may reveal rhonchal fremitus. Percussion resonance is heightened, from the chronic inflation of the lungs. Auscultation reveals harshness of the breath sounds, with greatly prolonged expiration, but frequently only adventitious sounds can be heard, namely, râles of all kinds—sonorous, sibilant, mucous, submucous, and crepitant. The râles are, as a rule, changeful, both in respect of their character and site, being much influenced by the act of coughing, especially the mucous variety. The expectoration varies much in respect of quantity. It is most usually frothy, partly fluid and partly viscid, and muco-purulent in composition. In a variety of the disease, commonly called bronchorrhœa, or pituitous catarrh, the expectoration is extremely abundant, overshadowing the other symptoms. It may be chiefly serous, and at the same time viscous, resembling white of an egg, or, on the other hand, it may be puriform. The subjects of this variety of bronchitis are often much debilitated and may emaciate rapidly. In still other cases, the expectoration becomes fœtid, which usually (though not invariably) indicates the presence of bronchiectasis. The sputum is considerably abundant, greyish or grey-green in colour, and on standing separates into three characteristic layers or strata. The uppermost of these resembles the frothy sputum of ordinary bronchitis; the middle, that of serous bronchorrhœa; while the lower stratum consists of a purulent matrix, containing small plug-like masses composed of fatty and granular material, various bacteria and decomposition products. These plugs are characteristic of this form of bronchitis; they were particularly described by Traube, who regarded the smaller bronchi as their place of origin.

We meet sometimes with cases which may properly be described as chronic capillary bronchitis. Usually, only a portion of lung is the seat of this lesion, but it may occur more or less generally. Fine clicking râles, with absence of dulness, constitute the main physical signs. There are in such cases great dyspnœa, and very marked cyanosis, if the condition be at all extensive.

As bronchitis advances, we find signs of over-distension of the right ventricle and increasing weakness of the heart, so that the circulation often signally fails, entailing venous congestion of the organs and tissues generally. These include albuminuria, dyspeptic troubles, signs of congestion of the liver, and the signs of pulmonary œdema, especially basic œdema, with dropsy of the feet and legs, and even of the trunk. Cyanosis also is present in great degree. While the obstruction in the lungs may entail cardiac failure, we have also to bear in mind that in many cases, seen at a stage more or less advanced, the bronchial affection may be dependent on long-standing mitral disease. Again, it may be remarked that in old persons there is a failure more or less of all the tissues from senile change, and this failure is greatly augmented by the de-oxygenated state of the blood. The heart in particular suffers in nutrition from this cause.

The question of the bodily temperature requires consideration. In many of the ordinary cases of bronchitis, no elevation of temperature may be met with, unless acute exacerbations occur. When persistent slight elevation is met with, especially in a comparatively young subject, the possibility of tuberculosis, or a tendency thereto, should be entertained. In the severe forms of the disease, with much inflammation in the tubes, the presence of cyanosis suffices to reduce what would otherwise be a pyrexial condition; even a subnormal temperature may be met with, owing to this maloxygenation of the blood, so throwing the observer off his guard. But pyrexia is commonly met with from time to time, and especially in the evenings, while in cases of fœtid bronchitis, hectic symptoms are frequently developed.

Diagnosis and prognosis.—But little requires to be said under this heading. The symptoms already described, when unattended by dulness and bronchial breathing in any region of the lungs, are absolutely unequivocal. Œdema of the bases will certainly give rise to some degree of dulness and to crepitation, but the distinctive signs of pneumonic consolidation are absent. This is entirely dependent on certain conditions. If any well-marked constitutional condition be present, especially gout or cardiac disease, the prognosis is unfavourable as regards complete recovery. Again, if the patient be in the lower ranks of life, his chances are unfavourable. Under all circumstances, the disease is most persistent, very prone to recur, and is rarely completely got rid of, unless prolonged careful treatment be adopted, or possibly residence in a warmer and drier climate. The emphysema is permanent.

Treatment.—Chronic bronchitis is met with under so many different conditions, and subject to so many modifying influences, that much thought and judgment are necessary in the matter of treatment. It is hardly necessary to say that warm clothing, great care in avoiding exposure to cold or sudden changes of atmosphere, and the maintenance of the highest possible level of general health, are elementary rules.

A primary duty is to investigate any constitutional defect, and rectify it as far as possible. This will include the general and special methods of treatment applicable to gout, rheumatism, tubercle, or syphilis, as the case may be.

When practicable, the patient should be sent to a spa, preferably in a dry mountainous region, and have a course of treatment suited to his condition. The sulphur waters are most valuable. Turkish baths should constitute a part of the treatment. If possible, the patient should be made to spend the winter (or, preferably, a few winters) abroad in a dry climate. A high altitude should be selected, if the kidneys are sound.

Attention to the state of the bowels is of great importance. Often a smart purge will cut short the progress of urgent symptoms, more especially excessive secretion attended by dyspnœa. Counter-irritants and stimulating frictions are often serviceable. In particular, the cautious application of croton-oil over a limited area, until a pustular eruption is produced, may be most efficacious in local exacerbations. Cod-liver oil is suitable for most cases, on account of its action as an expectorant as well as for its nutritive and restorative qualities. Exercises are most beneficial to sufferers from chronic bronchitis. They should be regulated so as to bring the muscles of the chest especially into play, and accompanied by the practice of prolonged inspirations and forcible expirations. In this way the circulation and nutrition of the bronchial tubes improve, the elasticity of the lungs is increased, and the secretion is moved outwards. The "compressed-air" cure, for the details of which special works may be consulted, acts much after the same fashion. It is often attended with considerable improvement, particularly when the pulmonary circulation is defective.

The drugs which are specially employed in bronchitis form a long list. It is best to do without them if possible, and persevere on the lines which have already been briefly indicated. Many of the so-called expectorants do little else than tickle the patient's œsophagus, and induce a fancy of well-being. The following indications, however, will be generally serviceable:—

When the cough is very harassing and expectoration scanty, inhalations of steam, or of ipecacuanha wine, diluted with thrice its bulk of water, may be given by the atomiser. In some cases, fairly large doses of opium may be temporarily given to subdue the cough. On the other hand, if expectoration is profuse, inhalations of terebene, or hot water to which a few drops of turpentine have been added, may be useful. Internally, the balsams of copaiba and tolu, alkalies, belladonna, tar water, sulphur, and the tincture of steel, are commonly to be relied on. When the expectoration is fœtid, tonics (iron, quinine, phosphorus) are imperatively called for; while inhalations, or intratracheal injections containing eucalyptus, creasote, or carbolic acid, must be persevered with.

Sometimes a striking and speedy improvement follows the use of iodide of potassium, when other measures have failed. It is not easy often to find the explanation of this, nor can any rules be laid down as to the cases suitable for its use. It is a drug which should certainly never be lost sight of in the treatment of any case of this disease.

Numerous symptoms and complications require attention in the course of chronic bronchitis, but little else can be done here than to indicate the most important of these. The state of the heart and circulation stands in the very forefront, and any indications of failure must be met with appropriate measures. For many cases the Schott baths and exercises will prove highly beneficial, gradually reducing the dilated ventricle and restoring the patient to comfort. Oertel's plan may benefit many cases. Should these methods be impracticable, a stimulating and tonic régime, rest in bed, and mild purgatives are the measures to be adopted. Strychnine, digitalis (cautiously), and carbonate of ammonium are the drugs to be relied upon. Cyanosis may require blood-letting and oxygen inhalations. Spasmodic dyspnœa is to be met with hot poultices, antispasmodics, as nitroglycerin, lobelia, opium, and purgation by mercury.

In order to allay the nervous disturbance and procure sleep, tepid baths and the occasional use of the bromides may be resorted to. As regards the employment of alcohol in chronic bronchitis, it must be laid down as a general rule that the younger the patient the less need is there at any time for resorting to it; and it should never be taken except under medical control, as in some of its forms it may be highly injurious. But, in the case of weak and elderly persons, some suitable stimulant is generally necessary, especially before going to bed, as it will be followed by a sense of comfort and the inclination to go to sleep.

FIBRINOUS OR PLASTIC BRONCHITIS.

A somewhat rare disease of the bronchi, consisting in the formation of fibrinous casts, which are expelled by coughing.

Etiology.—The disease is in all likelihood caused by a micro-organism; several organisms have been detected in the exudation, but nothing definite can be said about them. A distinctive organism has not been isolated. The predisposing conditions are uncertain, but the exciting causes appear to be much the same as in the case of ordinary bronchitis. Young males between 20 and 40 are most liable.

Morbid anatomy.—The white fibrinous casts may be deposited over a part, or over nearly the whole bronchial tree. They often form a beautiful picture of the bronchial system, even to the smallest ramifications. They consist of stratified layers of fibrin, containing mucous cells and blood corpuscles. The mucous membrane may be but little altered, and its epithelium intact. In other cases it is injected, and the mucous cells wholly or partially desquamated. In many respects the disease resembles fibrinous pneumonia.

Symptoms.—Fibrinous bronchitis occurs in an acute and likewise in a chronic form; but the latter is much the more common. Their symptoms are very much like those of the ordinary forms of bronchitis; in fact, the disease may be overlooked if the sputum be not examined for the characteristic casts, which are often small and obscured by mucus or blood. Dyspnœa is unduly prominent, owing to the blocking of the tubes. The chronic variety may last for many years. Hæmoptysis is not uncommon.

Diagnosis and prognosis.—This can only be made by the discovery of the fibrinous casts, which are hollow in the case of the larger tubes, and solid when formed in the smallest ones. In the acute form, this affection may be either mild or severe. If severe, a fatal issue is not uncommon from asphyxia, especially in the case of weakly persons. Sometimes the subjects of the disease are already phthisical. Speaking generally, recovery may be looked for in the course of two or three weeks. The chronic variety is rarely serious, and then only when complicated with emphysema, tuberculosis, or heart disease. In the majority of cases, recovery, though long delayed, will ultimately be brought about.

Treatment.—An efficacious treatment has not as yet been discovered. Biermer claims that the inhalation of atomised lime-water is highly curative. Sometimes an emetic is necessary to expel the membranes. The general principles of the treatment of bronchitis are, indeed, applicable to the fibrinous variety. Iodide of potassium should certainly be employed, and any constitutional tendency to disease rectified, as far as may be possible.

BRONCHIAL ASTHMA.

A neurosis, sometimes hereditary, sometimes acquired, manifesting itself in localised spasmodic contractions of the bronchi, and accompanied by more or less congestion of the bronchial mucosa.

Bronchial asthma has been recognised as a distinct malady for the last two centuries, previous to which it was mistaken with other forms of dyspnœa. Willis, v. Helmont, and Cullen first detected its individuality, and subsequently Laennec propounded its nature. Since Laennec's time, certain other views have been advocated as to its pathology, not without support; but the original view of Laennec holds the field at the present day, if anything, more securely than ever.

Etiology.—The disease respects no age, is in many instances undoubtedly hereditary, and is at least twice as frequent in males as it is in females. A good deal of confusion has arisen in regard to the causation of bronchial asthma. Thus some regard it as an expression of a morbid constitutional state, and it may indeed alternate with or accompany gout, rheumatism, and other states. Others have found a connection between asthma, neuralgia, dyspepsia, and epilepsy, suggesting it to be essentially a neurosis, or even a transformable neurosis. Others, again, have sought the explanation of the phenomena locally, in morbid conditions of the bronchial mucosa; while in not a few instances the disorder has been traced to nasal disease. Whatever constitutional or other conditions are found in association with asthma, it is undoubted that the disease is a neurosis; but it must be clearly observed that the causes are of wide range.

There is a large class of patients in whom the neurosis is manifested at an early age, and occurs, with but brief intermissions, over a considerable period, or even the whole of life. An asthmatic "habit" of body is produced—pale, nervous, and emaciated appearance, stunted growth, stooping posture, and rounded shoulders. The disease may then be likened to idiopathic epilepsy, which may pass off after a certain period of life, but which runs a definite course, and is but little influenced by treatment or external surroundings. In another class, the affection is lighted up at some time or other, owing to a cause or accumulation of causes which suffice to educe what may be a latent predisposition. In yet other cases, local peripherally-acting irritants produce the symptoms of the disease, which subside and may be completely removed on getting rid of the cause. Such cases correspond to other convulsive attacks which depend on reflexly-acting excitants, as apart from centrally situated sources of instability.

Of the factors which may precipitate an attack of asthma in predisposed persons, none are more notorious than certain conditions of climate. Thus, some who are much troubled in the country, get much better on removing to the interior of a large city. Many different idiosyncrasies are shown in respect of atmospheric causes; and in one variety of the disease termed hay asthma, or hay fever, the pollen of grasses regularly produces the affection each recurring summer. Reflex asthma may be induced by dyspepsia, pressure on the vagus, cold feet, uterine disorders, all sources of irritation of the bronchial mucous membrane, nasal polypi, and numerous other conditions.

Pathology.—Asthma is caused by an intense contraction of the muscular layer of the bronchial tubes over limited areas. This tonic spasm may arise with remarkable suddenness, persist for a longer or shorter period, and then vanish almost as quickly. It has nevertheless been supposed by certain observers that the constriction of the tubes is caused by a sudden vascular turgescence or "fluxionary hyperæmia" of the mucosa, with secretion of tough mucus. A third view is that tonic spasm of the diaphragm accounts for the attack. It would be out of place in a work like the present to discuss the various arguments for and against these theories. There is no doubt amongst pathologists at the present day that tonic spasm of the bronchial tubes does occur, and that it is present in asthma.

It is nevertheless true that, along with this spasm, there is a catarrhal element, often very marked. The advocates of spasm often overlooked this element, and no doubt failed to truly represent the morbid process. The sphincter-like constrictions of the tubes must of necessity cause congestion, by impeding the local venous circulation. And, moreover, such a congestion, frequently renewed, would ultimately result in a chronic catarrh of the mucosa, as is indeed found in cases of long-standing asthma. Certain bodies, called Leyden's crystals and Curschmann's spirals, have been found in the expectoration of asthma, and some have regarded them as causative irritants. There is no evidence to show that these bodies are produced before an attack of asthma, and, moreover, they are often found in certain cases of bronchitis. The Charcot-Leyden crystals are pointed octahedra, and are also found in leukæmia and in the semen. The spirals occur in two forms, and are composed of mucin. In one variety, they form twisted coils of concentrically arranged filaments; in the other, the filaments coil round a central longitudinal filament of remarkable brightness, which is generally supposed due to a peculiar transformation of the mucin. The eosinophiles are greatly increased in the blood in asthma.

In hay fever we have a tumefaction of the nasal mucosa. Particularly, the inferior turbinated body is swollen and obstructs nasal breathing. Here we have an irritant starting from the nose, and, proceeding along the Vidian and other nerves, begets a reflex asthma. This asthma is sometimes cured by scoring the turbinal bone with the galvano-cautery, and other local measures. On the other hand, the nasal symptoms may abate, and a congestive asthma arise in the bronchi. This is truly a vicarious asthma, and the irritant acts directly within the tubes on the fibres of the pneumogastric. The morbid changes which have been found within the lungs in long-standing cases are of a secondary character. Chronic congestion and thickening of the tubes (already referred to), emphysema, and dilatation of the right side of the heart, are the most constant conditions. No lesions have as yet been detected in the nervous system.

Symptoms.—Certain symptoms, mental, nervous, or dyspeptic, are often premonitory of an attack, and a source of warning to the sufferer or his friends. Then for a variable time, hours or even days, worrying symptoms may be present, such as cough, more or less dyspnœa, constriction in the chest, and general nervousness and lassitude. In a large number of cases, however, the paroxysm comes on quite suddenly, and especially during the hours of sleep, most commonly between 2 and 4 A.M. The intensity of the struggle which then ensues must be seen to be properly appreciated. It is essentially an overpowering dyspnœa. The chest is *fixed in the condition of inspiratory expansion*, the diaphragm is depressed, the abdominal muscles tense, and the accessory muscles of respiration are prominently brought into play.

The obstruction to the natural recoil of the lungs in expiration is brought about by the spasm of the tubes; they thus get distended with air, so we find that both inspiration and expiration are rendered most difficult: and expiration especially, as might be expected, is greatly prolonged. The persistent pulmonary inflation is the undoubted consequence of the bronchial constrictions, though various other theories have been advanced, which need not be discussed here. The action of the inspiratory muscles, including the diaphragm, is clearly directed to the utmost against the bronchial muscles, which are naturally antagonistic, but excessively so during the asthmatic attack. Such action on the part of the respiratory muscles is therefore compensatory, and not an additional element of spasm. The patient during the paroxysm assumes various positions, frequently the stooping posture, with the hands fixed to some support. There is an eager anxiety for abundance of air. The face assumes a pale hue, with lividity of the cheeks and lips, and other parts. The extremities get cold, and an abundant cold perspiration breaks out from the face and trunk. Respiration is noisy, the wheezing being audible for some distance. The paroxysms may terminate after a short time—half an hour to a few hours—with a scanty tenacious expectoration which does not amount to more than a few drachms, and may be less. In other cases, the attack goes on for days or over a week, getting gradually less severe. In some such cases the expectoration in the latter stages is considerable, and may give rise to what has beem termed humid asthma.

The physical signs consist of fixity of the chest, as already shown, but the intercostal spaces, suprasternal and supraclavicular fossæ, and epigastrium sink in on inspiration; percussion is hyper-resonant, and loud dry rhonchi and sibili, which completely mask the breath sounds over all affected areas, are heard on auscultation. The sounds are, however, very shifting, both in character and in site. The area of cardiac dulness is obscured or absent, and the hepatic dulness is obliterated. The temperature is never elevated, but is frequently subnormal. The urine is at first abundant, latterly scanty.

Diagnosis.—This is generally rendered easy by the striking characters which have been indicated. It is to be remembered, however, that a degree of bronchial asthma frequently complicates other affections, and especially acute bronchitis. Even the dyspnœa of cardiac and renal disease may be in part spasmodic. From laryngeal dyspnœa, bronchial asthma is easily distinguished. In the former there is stridor, and the dyspnœa is of the inspiratory type. The same applies to laryngeal paralysis, produced by thoracic aneurysm.

Prognosis.—Generally speaking, genuine asthma is but rarely completely got rid of, but a cure may be expected when some definite irritation provokes the disease, as a growth or other morbid condition in some part of the respiratory tract. The prognosis is best in youth, for if the disease be established in middle life, structural changes ensue of a permanent character. The actual paroxysm itself is never fatal.

Treatment.—The treatment of asthma by special drugs should only be resorted to during the paroxysm. Such drugs, with one exception, should not be used in the intervals of freedom.

The remedies to be used act on the nervous system or on the bronchial muscles themselves, and the following are principally to be relied on—opium, belladonna, iodide of potassium, nitroglycerin, or some preparation of nitrites (or fuming nitre papers), ethereal tincture of lobelia, and stramonium. Paraldehyde has also been recommended. Various combinations of these may be employed. In point of fact, the physician must find out what is most suitable to the case. Strong coffee and alcohol may prove serviceable as adjuncts. A mercurial purge is often highly beneficial. An emetic of ipecacuanha may give relief. The inhalation of chloroform may be cautiously adopted, and is always followed by temporary relief. The application of the induced current along the vagus in the neck may prove useful. The compressed-air chamber, in which the patient is made to inspire a compressed and therefore more highly oxygenated atmosphere, is often followed by great relief, which is in some measure owing to its powerful effect on the circulation.

In the intervals of the attacks, tonics, especially quinine, iron, strychnine, and arsenic, are to be administered. In addition, iodide of potassium should be thoroughly tried, and in this drug we have one notable exception to the rule which has been stated above. A prolonged course of iodide of potassium and arsenic may, in fact, greatly lessen the tendency to the attacks, or even effect a cure. If the right heart has become dilated, the Schott baths and exercises may be employed with great advantage.

In hay fever the most efficacious treatment consists in cauterisation of the hypertrophied mucous membrane. General and dietetic measures are highly important. A suitable climate, avoidance of exposure, warm clothing, and plenty of outdoor exercise, short of fatigue, are to be specially noted. The dietary should be liberal and yet carefully conducted. In some cases a special dietary has to be laid down, and if any constitutional tendency, as gout, be present, this is the more clearly necessary. A course of suitable waters at one or other of the mineral spas is to be highly commended, especially the sulphuretted springs. The Turkish bath should be used also, unless any cardiac condition prohibit it. Stimulants must only be taken under medical direction ; generally, whisky and water or dry sherry may be allowed.

BRONCHIECTASIS.

Permanent localised dilatations of the bronchial tubes.

The condition was first described by Laennec. Since then a great many observers have studied the subject, with various results. The Irish physicians Stokes and Corrigan, in 1837 and 1838 respectively, threw considerable light on the subject. In 1860, Biermer expounded in a lucid manner the chief causal agencies. The more recent writers, while adding little that is new, have contended variously for the importance or primacy of one or other of the recognised factors in the production of the disease.

Etiology.—Bronchiectasis depends for its production on the existence of other lesions of the lungs and bronchi, usually serious and of some duration, and which induce a weakness of the bronchial wall. No age is exempt, provided the predisposing lesions be set up. There is no satisfactory evidence to show that bronchiectasis ever arises primarily. No case of bronchiectasis is ever found post-mortem in which there is not bronchitis, or structural disease of the substance of the lungs, or both. But while the pulmonary lesion may so far determine the bronchiectasis, it is equally certain that both may advance together under the same impelling cause. Very rarely the condition is congenital. The lesions which chiefly induce bronchiectasis are—chronic bronchitis, chronic pneumonia, pleurisy, tubercle, stenosis of the bronchi, by aneurysm or tumour, and foreign bodies in the bronchi.

Morbid anatomy and pathology.—The dilatations of the bronchi are found in several forms, namely, uniform or cylindrical, saccular, moniliform (or resembling a row of beads), irregular or angular, and there are also mixed varieties. The lower lobes of the lungs are the most frequent seats of the disease. When the condition is unilateral, all the lobes are affected. The bronchial lesions themselves are of two kinds; the atrophic and the inflammatory. The former reduce the wall to a mere pellicle, consisting generally of the basement membrane and some connective or possibly muscular fibres. The inflammatory lesions are often peculiar. In some bronchi, papillary outgrowths, composed of granulation tissue, are formed, which may grow towards each other, coalescing at their apices, and so forming bridges stretching over parts of the bronchial wall. In other cases, where there is offensive secretion, the mucous membrane may become covered by squamous epithelium instead of the columnar, and a peculiar epithelial formation of lines and clusters is found in the deeper portions of the wall. In cases wherein a suppurating cavity is found, its bronchial origin may be revealed by a curious hypertrophy of muscular tissue in the thickened surroundings. By searching for some of these clues—the basement membrane, epithelial formations, or muscular remains—the histological diagnosis between a bronchiectatic cavity and a pulmonary excavation, long a matter of difficulty, can nearly always be made.

The foundation of the disease is laid by one or other of the conditions (already mentioned), which induce weakness of the bronchial wall, whether by inflammation or atrophy, or pressure on a bronchus, as by aneurysm. In the case of pressure, there is retention of secretion inducing inflammation of the bronchial wall, which gradually dilates under the weight of its contents. The foundation being laid, we have next clearly to keep in view the action of a dual set of respiratory forces. One force acts from within the tubes, consisting of the respired air. Another force acts outside the tubes, namely, the air in the pulmonary parenchyma. In the normal condition these forces balance each other, and in so far oppose each other. The respiratory force in the bronchi may be said to be always acting in the direction of bronchiectasis, while the tension of the air in the pulmonary vesicles is directed against the bronchial expansion. But if the forces in the pulmonary parenchyma should fail, the respiratory forces remaining normal in the bronchi, or if the latter be greatly in excess, then bronchiectasis will be liable to be set up.

What has just been said may be illustrated by common examples. When, for instance, a district of lung collapses and the finer bronchial tubes within the same become blocked, the inspired air will act on the bronchi distributed to the collapsed territory, but no reaction or support is given to their walls, which consequently will become dilated. Herein we find the dilated bronchi often resemble the fingers of a glove, with bulbous endings. Again, suppose a bronchus becomes constricted; the air beyond the stricture will accumulate, and the act of coughing will greatly augment the pressure of the air within the tubes. When a portion of lung undergoes cicatricial contraction, we have a cause acting in the same direction as the bronchial inspiratory force. The like is true of pleuritic adhesions. It is evident that if the bronchial wall be diseased, the distending forces will act with great facility. And it will also be seen that when a portion of lung undergoes atrophy, the pertaining bronchi will tend to expand, in order to fill up the vacancy, so to speak.

It has been alleged, and especially by Grainger Stewart, that the tissue of the bronchial wall may undergo a primary atrophy, which is unconnected either with the inflammatory process, or with the factors which induce concomitant atrophy of the pulmonary tissue, especially increased pressure of air. This is admittedly a theory, and no such isolated lesion has yet been sufficiently demonstrated post-mortem. At the same time, it is to be remarked that there is no reason why it should not occur, if only on the analogy of what takes place in other structures, and if it did, the inflammatory changes which would follow from retained secretion would obscure the original lesion.

Symptoms.—The symptoms of bronchiectasis are associated with the symptoms of various morbid conditions of the lungs, hence it is not easy to unravel the two, and frequently but little attempt is made to do so. With bronchiectasis there is always paroxysmal cough, and usually there is profuse expectoration, easily brought up, and at first muco-purulent in character. It is this paroxysmal coughing up of large quantities of secretion which is the most reliable indication of bronchiectasis. In the intervals, cough may be slight or absent. An attack may often be induced by changing the position of the patient. The sputum shows different strata on standing, a granular detritus being at the bottom and a muco-purulent fluid above. It should be examined for elastic tissue, and if areolæ or fragments of these are found, it shows that there is ulceration of the lung, and this may serve to clear up difficult cases. As the case advances, the sputum becomes fœtid. This is highly characteristic of the disease. Hæmoptysis is frequently met with, which may vary from a few streaks to a fatal discharge. Dyspnœa is nearly always present, especially great on exertion, and depends much on the pulmonary lesions present.

Inspection of the chest shows either distension, local or general, if emphysema be present, or retraction, in cases of chronic pneumonia. Percussion also will be variable. But over a bronchiectasis or bronchiectatic cavity, which is near the surface, it is tympanitic, amphoric, or cracked-pot. Auscultation discloses râles which are unusually large, especially over such situations as the edge or base of the lung. In considerable dilatations, tubular or cavernous respiration is heard. In short, we have the usual signs of pulmonary cavities, with bronchophony or pectoriloquy. The progress of bronchiectasis is characterised by digestive derangements, intermitting albuminuria, and circulatory disturbances. The bodily nutrition is often wonderfully sustained, though the aspect is cachectic and cyanotic. Latterly, there are usually emaciation and intermittent febrile attacks. Abscess of the brain sometimes occurs.

Diagnosis.—It is clear from the foregoing that the only positive sign of a bronchiectasis is the sign of a cavity in the lung, and the question therefore to be settled is as to the nature of the cavity. The expectoration may give much help in this investigation, and its characters have been already noted. In fœtid bronchitis the expectoration may be similar, but there is absence of the characteristic paroxysms and of the physical signs of a cavity or cavities. The sputum in pulmonary gangrene has generally a characteristic fœtor, but, in addition, the history of the case and the severity of the constitutional symptoms will afford a guide. Still, gangrene may be a result of bronchiectasis. From tuberculous excavation, a bronchiectasis may generally be diagnosed by the frequent absence of dulness surrounding it, and this condition is perhaps to some extent productive of another diagnostic sign, namely, the more rasping character of the cavernous breathing in the bronchiectatic cavity. The sounds also are more diffused than in the case of a cavity situated in dense tissue. When the bronchiectasis is in the upper lobe, it is very commonly placed near the sternum; a bronchiectatic cavity is rare at the apex. Again, bronchiectasis is most frequent in the base, and the concomitant pulmonary changes tend to extend upwards. The size of the cavity in bronchiectasis generally remains stationary, in phthisis it enlarges. The filling and emptying of the bronchiectatic cavity will yield their respective physical signs. Of course the sputum should be examined for tubercle bacilli, or areolæ of elastic fibres. In the case of a limited empyema, discharging through a bronchus, there will be pneumothorax, while the strictly purulent character of the sputum is also distinctive. The physical signs in bronchiectasis are very changeful.

Prognosis. — Bronchiectasis is undoubtedly a serious condition; it nevertheless may have a very long duration, and the whole course of the disease depends on its extent and the general condition. The establishment of bronchiectasis points to a grave failure. Cases may so far heal by cicatricial changes, while the adoption of suitable remedial measures, medical or surgical, may be followed by a great amelioration of the symptoms.

Treatment.—The various indications already given in the treatment of chronic bronchitis are more or less applicable in the case of bronchiectasis, especially the practice of forcible expirations with simultaneous compression of the chest. In this way the secretion is moved outwards. The use of astringent, tonic, and antiseptic drugs is necessary. Iron, quinine, strychnine, and balsams may be given internally when expectoration is profuse. Cod-liver oil should also be perseveringly pushed, if at all practicable. Such agents as tar, creasote, carbolic acid, or eucalyptus are given both by the stomach and directly by the atomiser. The creasote chamber is a powerful and efficacious, though rather trying, process, and will nearly always effect much good. In more recent years, a method which promises to be of service has been revived, and consists in the administration of suitable drugs by intratracheal injection. In particular, Grainger Stewart and Gibson have advocated this plan, and they recommend the injection twice daily of a drachm of the following mixture:—Menthol, 10 parts; guaiacol, 2 parts; and olive oil, 88 parts. A special syringe is used, and the laryngoscope is unnecessary. The following are the directions:—"The patient thrusts forward the tongue, grasps it and holds it there. The nozzle of the syringe is directed over the back of the tongue, care being taken to avoid touching it or the palate, and carried backwards until the tongue and epiglottis are passed. The point is then directed downwards and slightly forwards, the instrument being so curved that, with the handle against the upper teeth, the point is in the glottis. The injection is then rapidly administered and the instrument withdrawn." Some have recommended the injection of antiseptics directly into the cavities through the chest wall.

Should the methods of inhalation and intratracheal injection fail, another means, and one which will surely be the more perfected as time goes on, falls to be considered in suitable cases, namely, a surgical operation. This is especially indicated where a large-sized cavity is present in one lung, though some have advocated it when there is such a cavity in each lung. Having determined by the use of the aspirating needle the exact location of the cavity, a free incision, often with resection of a portion of the ribs, is made, and the contents evacuated by drainage and irrigation. The pleura, if not already adherent, must be secured to the thoracic parietes. The various details of the operation will be found in appropriate surgical works.

STENOSIS OF THE TRACHEA AND BRONCHI.

A chronic constriction of the lumen of the trachea or of a bronchus.

Etiology.—Although in bronchial asthma we have, in point of fact, a constriction of the bronchi, yet the condition termed stenosis is applied to such obstructions as are caused (*a*) from without, compression of the air-tube, as by an aneurysm, mediastinal tumours, enlarged glands, or goitre: or (*b*) from within, constriction of the air-tube, in the form of cicatricial contractions, new growths, and chronic hypertrophies of the membrane. The presence of foreign bodies will also produce a stenosis of the air passage.

Morbid anatomy.—Cicatricial contraction may be brought about by syphilitic disease, especially in the trachea, preceded by granulation tissue growth. Carcinoma usually causes compression, either at the glands, or as it proceeds along the lymphatics which lie outside the tubes. It also infiltrates the wall, and even the lumen of the tubes. A foreign body may be impacted in the tube and surrounded by new growth of tissue. When compression is brought about by aneurysm, the bronchial wall gets eaten away.

Symptoms.—Stenosis of the trachea is characterised by an active and painful sense of tightness or constriction in the chest, and by constant dyspnœa, which is chiefly inspiratory in type. This may be extreme, and cause action of the accessory muscles and retraction of the more yielding parts of the chest. There is also a harsh metallic cough.

In bronchial stenosis, if the tube involved be large, there is also dyspnœa, hard cough, and often expectoration. In severe cases there is cyanosis.

Diagnosis and prognosis.—In the case of tracheal stenosis, the laryngoscope may clear up the case. If the obstruction be in the larynx, the laryngeal movement is exaggerated, and the head inclines backwards, while in obstruction of the trachea, laryngeal movement is slight or absent, and the head inclines forwards; also, in cases of some standing, the upper part of the thorax falls in. Whether tracheal or bronchial, the stenosis gives rise to a stridulous condition of breathing, which may be audible for some distance, and the sibilant or piping sound is heard on auscultating over the obstruction. This is always serious, except in the case of tumours of the neck, as goitre. Syphilitic constriction is somewhat more hopeful.

Treatment.—Inhalations of oxygen or chloroform may relieve the dyspnœa. In the case of cicatricial constriction, large doses of iodide of potassium and mercurial treatment should be adopted, as the condition is most commonly syphilitic. If ulceration be present, topical applications may be applied by injection or insufflation. Surgical interference, as by dilatation of the stricture, may be suitable for some cases. If dyspnœa threaten life, tracheotomy has to be resorted to. In bronchial stenosis, little besides palliative measures can be attempted. It is probable, however, that the surgery of the future will successfully deal with some of these cases.

FOREIGN BODIES IN THE AIR PASSAGES.

During a deep inspiration, any substance in the mouth, especially if in the neighbourhood of the fauces, is apt to be sucked into the larynx. In this way particles of food, and foreign bodies of all sorts, are accidentally introduced. In bulbar and post-diphtheritic paralysis, the same thing is apt to occur. The foreign body may remain in the larynx, or pass on to the trachea, or a bronchus. If in the trachea, it may remain there, but generally passes to the *right* bronchial tube. This is owing to the anatomical conditions at the bifurcation, and the larger size of the right bronchus. The foreign body will ultimately lodge in a bronchus, which, in the inspiratory expansion, will suffice for its accommodation. If the body be round and smooth, collapse of the pertaining portion of the lung will ensue, while, if the body be irregular, admitting a certain proportion of air to pass, the dependent portion of lung will become emphysematous.

In many cases, wherever situated, the foreign body will remain for an indefinite period, doing no harm, and a capsule of connective tissue will grow around it. In other cases a local inflammation will be produced. If in a bronchus, pneumonia, abscess, or even gangrene may be set up in the pulmonary tissue. This "foreign body pneumonia," as it has been called, is very liable to undergo purulent change, and if free evacuation be not possible for the retained secretions, they may become fœtid (or gangrenous), and infect other portions of the lung, and thus an entire lung, or even (from insufflation of the irritating substances) both lungs, may become the seat of patchy pneumonias, riddled with cavities, and be destroyed.

Similar conditions may be produced by foreign substances gaining admission through perforations of the air passages, as in the case of a communication with the œsophagus, and the insufflation of food into the bronchi.

The entrance of a foreign body into the glottis, trachea, or bronchus, will usually be indicated by sudden suffocative spasm and cough. In the case of the glottis, sudden death may ensue. The physical signs are those of obstructed respiration, feeble respiratory murmur, general or unilateral, with sibili or rhonchi.

Expulsion of the foreign body may rarely be accomplished if the patient be inverted, and smartly struck between the shoulders. Spontaneous expulsion occasionally occurs on coughing, even after the lapse of a considerable time. If the body is in the upper part of the larynx, it may sometimes be extracted from within. In most cases, however, tracheotomy is required, and the body extracted by suitable forceps. This can generally also be accomplished if the obstruction be in the trachea or main bronchus.

A. G. AULD.

DISEASES OF THE LUNGS

PULMONARY CONGESTION AND ŒDEMA.

UNDER this description are included several conditions of hyperæmia of the lungs, which may be accompanied by transudation of fluid from the vessels into the air spaces and interstices of the lung.

History.—Some uncertainty exists as to the limitations of the description, and the name is not used by all physicians in quite the same sense. More particularly, the significance of "congestion" has varied. By the majority this term has been used to imply a condition of pulmonary hyperæmia, determinable by a variety of factors. Others have described under "acute congestion" a definite disease with distinct symptomatology. The symptoms, however, which they describe closely resemble those of slight or abortive cases of acute pneumonia. It is open to question whether such a group can be fairly separated. In practice, the attempt has been found productive of confusion, "congestion of the lungs" being too often used vaguely to include pneumonia and other inflammatory conditions. The more generally accepted use of the term will be adopted in the present work.

Etiology.—It is advisable to distinguish (*a*) active congestion, (*b*) passive congestion, (*c*) œdema, which may accompany either of the preceding.

Active congestion.—It is not so easy as it might be supposed to separate active (or acute) congestion from passive. Active congestion occurs as an accompaniment of certain inflammatory processes, as for example bronchitis, pleurisy, tuberculosis. It is an essential stage in the development of acute pneumonia, and this has perhaps given rise to the difficulty in classification to which reference has been made. Doubtless some cases of pneumonia run a modified course, aborting at the stage of congestion, but to be classed nevertheless as pneumonia. Apart from this, congestion may be determined by prolonged exposure, especially among alcoholic and gouty subjects, and is usually an extension from or complication of some degree of bronchitis. It may also be induced by severe effort, and probably by the inhalation of unduly warm or cold air and irritant vapours or gases.

Passive congestion.—This is the expression of stasis of the circulation, dependent on physical conditions which may usually be traced. It is to be looked for in mitral incompetence or stenosis with insufficient compensation, and in dilatation of the left heart from different causes. It may result from the pressure of tumours on the pulmonary veins. It is described as hypostatic congestion, when it occurs towards the posterior aspect of the bases of the lungs, in patients compelled to maintain the recumbent posture for a lengthened period with enfeebled circulation, as in many of the fevers. It occurs sometimes in relation to cerebral apoplexy, the congestion being most evident on the hemiplegic side. It may result from poisoning by certain drugs, *e.g.* morphine.

Œdema.—Transudation of fluid follows congestion, whether active or passive, when the congestion is considerable. Thus a certain amount of œdema is frequently associated with croupous and catarrhal pneumonia and other inflammatory processes in the lungs. Œdema occurs characteristically in the presence of prolonged stasis of the pulmonary circulation, as in mitral disease or cardiac enfeeblement from other causes. In this sense it occurs in aggravated form shortly before death, in relation to fevers and a variety of conditions—the affection described by some physicians as *pneumonia serosa.* It may be part expression of more distant circulatory failure, along with other evidence of anasarca, as for example in nephritis. Or it may result from local circulatory interference, as by new growths. Lastly, it doubtless results from aspiration exerted in the alveoli during inspiration, when some cause of obstruction to the entrance of air exists in the respiratory passages. In studying œdema, regard must be had, not only to the more evident mechanical cause, but also to disturbing influences in respect of the innervation and nutrition of the pulmonary vessels.

Morbid anatomy.—In the stage of congestion the lungs are evidently over-filled with blood, which may be bright red or venous in hue, according as the congestion is active or passive. When the latter condition has been long continued, the lung assumes the characters of "brown induration." The organ is bulky, and unduly resistant in whole or part, and of a reddish brown colour. Microscopic examination shows the capillaries to be distended with blood, and the alveoli to contain shed epithelium, blood cells, and pigment. In œdema, the bulky lung feels like a sponge half-full of water. The frothy albuminous liquid, of yellow or reddish tinge, contains blood and epithelial cells in varying number. It infiltrates the air spaces, bronchioles, and even the bronchi as well as the interstitial tissue. The condition is usually bilateral, but is often more pronounced on one side than the other. Along with the changes in the lungs, there are determinable changes in the heart or other portion of the circulation, according to the nature of the case. Hæmorrhagic infarcts of the lung may result from the congestion. The appearances presented by these are described in relation to Pulmonary Embolism.

Symptoms.—**General features.**—In slighter degrees there may be no determinable symptoms. The condition will naturally be suspected in all cases of cardiac enfeeblement or other circulatory failure, of which it forms an important expression. Most obviously, there will be increased rate and shallowness of breathing. The difficulty of respiration may amount to orthopnœa. The face tends to become livid. There may be actual cyanosis, with engorgement of the jugular veins and projection of the eyeballs. In children, convulsions may supervene. Cough is troublesome. The patient discharges at first a frothy, perhaps blood-stained, sputum. Later, when œdema is pronounced, the expectoration becomes abundant and watery. More considerable hæmoptysis may occur. The symptoms of active congestion and œdema are similar to those of the early stage of acute pneumonia. As has been already indicated, there seems reason to believe that a group of cases described by some under the name acute congestion, ushered in by chill, followed by pyrexia and evidence of lung implication, are really pneumonic in type.

Physical signs.—Limitation of the respiratory excursion may be noted. Percussion may reveal at first a certain degree of hyper-resonance, and this may continue even after some degree of œdema is present. With advancing œdema dulness supervenes. Frequently dulness is determinable in one area, perhaps the base posteriorly, along with hyper-resonance above or anteriorly. The respiratory murmur is generally feebler but harsher, and is accompanied or masked by crepitations—fine, medium, or coarse—in different cases. Corroborative physical evidence may be obtained by examination of the heart and vessels.

Diagnosis and prognosis.—At the stage of congestion this is apt to be vague. The diagnosis of œdema is of especial moment, and rests on the determination of the physical signs which have been described, in presence of circulatory failure, either from heart affection or from other cause. The diagnosis of active congestion is to be given with caution. In all such cases, pneumonia and other inflammatory affections must be carefully excluded. This depends on the nature and removability of the causal factor. The existence of pulmonary œdema points to grave disturbance of the circulation, and, as has been indicated, frequently ushers in the inevitable end (hypostatic congestion). On the other hand, when the circulatory failure can be corrected or compensated, œdema, even large in amount, will speedily disappear. Œdema complicating an acute inflammatory attack, affects the prognosis unfavourably.

Treatment.—This is ultimately the treatment of the causal condition, whether acute or chronic. It involves a careful consideration of the circulatory disturbance. The therapeutic measures to be adopted in each case are discussed under their appropriate heads. Great benefit is often derived from immediate blood-letting, either by venesection, cupping, or leeching. Stimulating applications, such as turpentine stupes, to the chest are of value. Internally, cardiac tonics and alcoholic stimulants will be found generally helpful. In urgent cases the subcutaneous injection of ether or camphor must be employed. Purgatives, diuretics, and diaphoretics may be of service, the latter being particularly indicated in cases of more active congestion. Of purgatives, mercury and concentrated salines are the most efficacious.

PULMONARY EMBOLISM AND THROMBOSIS.

Pulmonary embolism consists in the plugging of the pulmonary artery, or one of its divisions, by a clot or other foreign matter carried from a distance; pulmonary thrombosis, in the blocking of the vessel by a clot formed *in situ;* the clinical phenomena varying as the plugged vessel is large or small and as the plug is simple or infective.

History.—The condition was imperfectly understood until the elaboration of the subject by Virchow. This has been confirmed in most part, and little added to, by subsequent observers. The carriage of emboli from the systemic veins and the right side of the heart, and their impaction in the pulmonary artery, are well recognised, as is the occurrence of thrombosis in the pulmonary artery. But difference of opinion and some uncertainty surround the question of the relationship between pulmonary embolism and hæmorrhagic infarction of the lung. Several points, to which reference will be made, remain to be cleared up.

Etiology.—The plug which forms the pulmonary embolus may be derived from the systemic veins, or the right heart. In the case of the systemic veins, it may result from injury, inflammation, suppuration, or marasmus, following some general affection. The puerperium bears an important causal relationship. Fat embolism is described in case of fracture of the long bones. In the case of the right heart, coagulation is likely to occur under conditions which conduce towards reduction in the rate of flow. Such coagulation occurs especially in the right auricle, in case of cardiac enfeeblement from any cause, and is hastened by malnutrition.

With regard to effects, much will depend on the nature of the embolus, whether it be simple or infective. If simple, the effects vary according to the point at which the pulmonary artery is plugged. If a small division be implicated, the results are slight, while if the main stems be blocked, the effects are rapid and most grave. Death may occur immediately, or be delayed for a varying interval of time. When immediate, the fatal event is to be explained as due to the sudden occlusion of the main trunk or a leading branch of the pulmonary artery, and consequent asphyxia, with resultant circulatory and respiratory paralysis. By some it has been attributed to the induction of cerebral anæmia—the heart stopping in diastole. By others it has been traced vaguely to nervous shock. When death occurs later, it may be due to an extension of coagulation, or to further embolic impaction. The initial diminution in the gravity of the symptoms which may be witnessed and their recurrence would seem to justify this view. Recovery, which seldom results in grave cases, has been traced to compensatory dilatation of other branches of the pulmonary artery.

Hæmorrhagic infarction of the lung is frequently associated with embolism, but the exact relationship is not established. On the one hand, hæmorrhagic infarction may be found without traceable embolism, and, on the other, embolism of a portion of the pulmonary artery may be determined without evidence of an infarct. If the embolus be infective, metastatic processes will be established in the lung, their nature depending on that of the primary source of the embolus. Thrombosis is the expression of stagnation of the pulmonary circulation. Such stasis may be due to general or local causes. Three factors may be discussed as contributory, namely, an altered condition of the blood, enfeeblement of the right heart, and disturbed nutrition of the pulmonary vessels.

Morbid anatomy.—When recent, the embolus or thrombosis may be discovered as a more or less firm coagulum in the trunk or a branch of the pulmonary artery. Where a branch is involved, the presence of an accompanying infarct will guide the search. Later on, the coagulum is absorbed in large part, and may be traced merely as fine filaments in the lumen, or as an irregularity on the wall of the vessel.

An hæmorrhagic infarct is not constantly present, and, as already stated, its presence does not predicate embolism. The appearance of an hæmorrhagic infarct varies according to its age. In shape it is conical, the apex being directed towards the root of the lung, and the base being subpleural. It may involve the larger part of a lobe, or be no larger than a pea. It is commonly situated in the lower part of the lung. When fresh, it is of dark red colour and moist. In a few days it becomes friable, and gradually loses its colour, presenting the appearance of a pale area, surrounded by an inflammatory zone. Occasionally necrosis or gangrene or caseation follows. Still later, nothing may be traceable save a scar, more or less pigmented. The pleura over the infarct is at first natural, but rapidly loses its glistening character, and becomes injected and perhaps covered by fibrin. When cicatrisation results, some degree of puckering and thickening of the pleura may be evident. If the embolism be of infective nature, abscess formation or gangrene or pneumonia may be present. The appearances of these conditions are considered elsewhere.

Symptoms.—The clinical picture is not a constant one. In some instances, the symptoms are so slight that the diagnosis may not be made or even suspected. In others, death occurs so quickly that the patient is not seen in life, and the diagnosis rests on the correct interpretation of the report of the patient's friends or nurse. A distinction must be drawn between simple and infective cases.

Simple.—It is convenient to divide the simple cases into two chief groups, namely, severe and slight.

Severe or acute.—The patient, perhaps in the puerperium, or during treatment for phthisis or typhoid, is suddenly seized with extreme dyspnœa, and falls into a syncopal state. The syncope may be disturbed by convulsive paroxysms, and the patient may die before medical aid is obtained. Sometimes death seems instantaneous. The asphyxial and syncopal phenomena are usually present together, but either may be the more prominent. Short of sudden death, dyspnœa is likely the leading feature, with less or more syncopal tendency. Consciousness may be retained, and the patient gives expression to his sense of impending death or to pain which he experiences. The pain, which, however, is not always present, may be referred to a particular area of the chest. There is generally little or no cough. The respiration becomes more and more embarrassed, perhaps irregular or intermittent. The surface of the body becomes livid, cold, and clammy. The pulse is feeble and uncertain, and finally ceases. Where the condition is further prolonged, a reactionary pyrexia of three or four degrees often ensues. Final recovery is exceptional. Nevertheless a fair number of well-authenticated instances is on record where, after symptoms of the gravest sort, death did not follow.

Apart from evidence of alteration in the respiration and pulse, which have been considered, the physical signs are of little import, and there is usually little time to look for them. Diminished excursion of the chest, with enfeeblement or accentuation and perhaps some harshness of the respiratory murmur, may be noted. Increase in cardiac dulness towards the right may sometimes be determined, and murmurs over the pulmonary artery. The signs may be inseparable from those of the precedent condition, *e.g.* pulmonary tuberculosis.

Slighter cases.—The symptoms are the same in kind, but much less intense. Varying degrees of dyspnœa, palpitation, with corresponding irregularity of pulse, pain in the side, and cough, with expectoration of blood-stained sputum or of pure blood, may all be noted. Naturally the accident will be anticipated under certain conditions of the circulation and lungs, but it may be difficult to discriminate between symptoms or signs referable to the precedent condition and those clearly referable to embolism or thrombosis. Thus pyrexia, which, as already stated, may occur as the effect of embolism, is a symptom which may be interpreted differently by different observers. Where a pulmonary infarct occurs, some degree of pleurisy is often present, and pleural effusion not uncommonly. The physical signs, which are not always definite, may include limitation of the excursion, perhaps percussion dulness in case of infarction, and either enfeeblement of the respiration, or sometimes bronchial breathing and increase of vocal resonance. Coarse crepitations, limited in distribution, may perhaps be noted. Where the lung has been previously implicated, it is easy to go wrong in the interpretation of physical signs.

Infective.—When the embolus is of infective nature, there may follow abscess formation or a septic pneumonia. The condition will be ushered in by rigors and pyrexic disturbance, and the symptoms will merge into those of abscess or pneumonia, or form accessory features of a septicæmia, which has been, or will be, diagnosed on other grounds.

Diagnosis.—The physician must be alive to the possibility of the occurrence of embolism in presence of certain precedent conditions. These are especially the puerperal state, phthisis, marked enfeeblement of heart from various causes, and thrombosis of systemic veins. Stress is to be laid especially on the sudden onset of otherwise unaccountable dyspnœa, with or without syncopal seizure and convulsions. In slighter cases, the occurrence and continuance of unexplained blood-stained expectoration may be significant. It is necessary to be careful that all other causes of urgent dyspnœa, as pneumothorax in phthisis and cardiac embarrassment, be excluded. This will generally be possible by a consideration of the physical signs, along with the clinical history of the patient. In septic conditions, the local phenomena are usually merged in the general diagnosis.

Prognosis.—In acute cases the prognosis is unfavourable. Indeed, the fatal issue may have occurred before the question of prognosis is possible. In other cases, the prognosis depends chiefly on the causal and associated conditions. In infective varieties, the outlook is extremely bad.

Treatment.—This divides itself naturally into prophylactic and remedial.

Prophylactic.—The most important indication is complete rest, in presence of any of the causal conditions to which reference has been made. The woman after parturition, the patient with thrombi in the veins of his extremities, the subject of circulatory enfeeblement, must, in addition to all special treatment, have rest. The measure of rest will depend on the characters of the individual case. In some instances it may amount to continuance in the recumbent posture for weeks, with avoidance of all effort. Even the natural effort of defæcation may be advantageously aided by enema. In less urgent conditions, and later on in all, frequent resting on a couch throughout the day is to be enjoined.

Remedial.—This is largely symptomatic and palliative. Much depends on the gravity of the case. In the more acute there may be no opportunity afforded for treatment. When this is possible, the best effects may be expected from the use of morphine in full doses, preferably administered subcutaneously for the sake of expedition. Ether, by inhalation or subcutaneously, or other stimulant, *e.g.* caffeine, camphor, alcohol, may stave off the fatal issue. Thereafter, and in slighter cases, a careful study of the symptoms and the causal condition will suggest appropriate palliative measures. It must be borne in mind that the same causes which have induced the first accident may determine a second, and hence remedial measures are to be pursued hand in hand with prophylactic.

PULMONARY HÆMORRHAGE

Pulmonary hæmorrhage occurs in relation to a number of conditions. Some of these are not primarily pulmonary. Reference is made to such under their appropriate headings. But it is convenient from the clinical standpoint to group those conditions which have one feature in common, namely, expectoration of blood or hæmoptysis.

Hæmoptysis is the coughing or expectoration of blood which proceeds from leakage in the vessels of the lungs or respiratory passages, the leakage being produced by a variety of causes.

History.—From the earliest times hæmoptysis has occupied a conspicuous place in clinical medicine, on account of its important relationship with pulmonary tuberculosis (phthisis). Prior to the discovery of the tubercle bacillus, much discussion took place regarding the production of phthisis by hæmorrhage (*phthisis ab hæmoptoë*), and a good deal of apparent evidence was advanced in support of the view that phthisis might be thus induced. Recently, the consensus of opinion supports the belief that hæmorrhăge from the lungs, when related to phthisis, is to be interpreted as an effect rather than a cause. A further source of much discussion has been the connection between hæmorrhagic infarct and pulmonary embolism and thrombosis, and reference is made to this under the latter. On this subject the last word has not yet been spoken.

Etiology.—The blood may proceed from either the respiratory passages or the lungs proper. Following Laennec, some observers still divide the condition into (*a*) bronchial, (*b*) pulmonary hæmorrhage. This serves no useful purpose, and is apt to prove embarrassing, if not misleading. We recognise that hæmoptysis may occur in relation to morbid conditions of blood, as in pernicious anæmia, hæmophilia, scorbutus, purpura, pulmonary embolism or thrombosis; morbid conditions of blood vessels. These, which may be associated with and dependent on morbid blood conditions, include congestive, atheromatous, suppurative, ulcerative, and gangrenous processes, new formations (tubercle, cancer), and parasites (hydatid within the lung, or the *Distomum Westermanni* within the bronchial tubes—endemic hæmoptysis), or they may result from direct injury. Such morbid conditions may be primary in the lungs, *e.g.* tuberculosis, croupous pneumonia, or may be the expression of more distant affections, *e.g.* pulmonary congestion dependent on mitral disease, more particularly stenosis. Vicarious bleeding from the lungs must be admitted as occasionally occurring in connection with menstrual irregularity. Recurrent hæmoptysis has been described in elderly subjects, apart from obvious lung implication.

Hæmoptysis, as seen most frequently, is produced by capillary oozing, or the rupture of small vascular twigs. When blood is discharged in large amount, it probably results either from ulceration into a branch of the pulmonary artery of considerable size, or from rupture of an aneurysmal dilatation of such a branch, or an aneurysm from without into the respiratory passages. The immediate cause of the bleeding varies. There may be no obvious reason, but it can sometimes be traced to excitement, sudden effort, more particularly the lifting of heavy weights, cough (as in whooping-cough), a large meal, the use of alcoholic and other stimulants. Sudden chill, as by a draught of cold fluid, has been found effective, doubtless by the rapid alteration of blood pressure. Similarly, hæmoptysis has apparently been determined by cerebral injury. Certain other etiological aspects of hæmoptysis are described under the different diseases with which they are associated.

Morbid anatomy.—The anatomical appearances are various, according to the cause of the hæmoptysis. The consideration of these is fully entered on under these different conditions, and is therefore passed over here. From whatever cause, the blood escapes either from capillaries or from arteries and veins. In the former instance, it is not easy to determine whether this occurs by rupture or diapedesis. In the case of larger vessels, rupture occurs as the result of any of the causes which have been considered already. In the disintegrative stages of tuberculosis, rupture is favoured by the lack of support afforded to the vessel on the side of a vomica. Minute aneurysms occur in such cases, and are prone to give way. It is particularly from aneurysms of this class that the larger escapes occur. In veins the accident must be sharp and sudden, if hæmorrhage be induced. Otherwise thrombosis is more likely to result.

The appearance of the lung is various, according to the extent of the hæmorrhage. Where a hæmorrhagic infarct has occurred, the features are characteristic, and are described under pulmonary embolism and thrombosis. When blood has invaded the lung freely, as by the bursting of one of the aneurysms referred to, the vomica or vomicæ and smaller bronchi are found filled with fluid or clotted blood, the larger bronchi and trachea with more frothy blood. Large areas of lung tissue—away from the point of rupture—may be charged with blood, which has been aspirated. The rest of the lung, if the hæmorrhage has been considerable, will be conspicuously pale. If death does not occur for some time, there may be little anatomical evidence of the bleeding, as the corpuscular elements are quickly absorbed. The conception of a *phthisis ab hæmoptoë*—happily now discarded—had little post-mortem corroboration. It may be said with certainty that pulmonary tuberculosis, which seems to follow a hæmoptysis, has been really precedent, while it may equally be affirmed that, granted the existence of a limited tuberculous focus, the disease may be rapidly distributed by the washing away of bacilli in the blood current and their aspiration into previously unaffected portions of lung.

Symptoms.—The bleeding may take place quite suddenly in less or greater quantity. Sometimes, however, premonitory indications are present. The patient may complain of a feeling of oppression about the chest, or of palpitation. More particularly is this the case with patients who have already experienced hæmoptysis. It may be that their very anxiety is a cause of the occurrence. In general, the onset is unexpected. A tickling sensation, accompanied by warmth about the back of the throat and a peculiar saltish taste, is often remarked. The patient feels he must cough, and blood is expectorated. At other times, the discharge of blood is overwhelmingly rapid. It wells from mouth and nose. There may be intense dyspnœa, amounting even to asphyxia, with conspicuous cyanosis.

The character of the sanguineous expectoration is various. There may be but a slight tinging of mucous or muco-purulent discharge, or pure blood may be ejected, usually more or less frothy, either bright red, or more frequently dark in colour, and of alkaline reaction. It is sometimes clotted, or takes the form of bronchial casts. The quantity is also variable, from an inappreciable amount to a basinful; indeed, to the maximum that can be lost short of death—for death seldom results directly from the hæmoptysis. The duration of symptoms is different in different cases. Sometimes an almost constant oozing continues for days, or, with longer intervals, for weeks. In other cases the hæmorrhage is large and soon over, to be repeated in a few hours or days or weeks. For some time after the principal discharge, the expectoration, *e.g.* of a tuberculous patient, remains tinged, or clots may be passed from time to time, and occasionally bronchial casts.

Accompanying a serious hæmorrhage there is more or less constitutional disturbance. More particularly, there is circulatory excitement, the pulse being increased in rate, full and bounding, but easily compressible. Following the discharge, some degree of pyrexia may be noted and evidence of more or less pronounced anæmia, with coldness and clamminess of the extremities. Death is not a common termination, even when the hæmoptysis is alarming in degree. It may supervene, however, either by asphyxia from the flooding of the bronchial passages; or by anæmia, from the actual quantity of blood lost. This is not a condition when the determination of physical signs is of importance. The lungs will in most cases not require examination, nor the state of the patient warrant it. The presence of blood in the alveoli or vomicæ and bronchial passages may, however, be revealed by crepitations, which vary in character according to the amount and situation of the blood.

Diagnosis.—This includes, first, the determination that the blood comes from the respiratory passages directly, and, second, the determination of the seat and cause of the bleeding.

In the first place, bleeding from the nose, mouth, naso-pharynx, œsophagus, and stomach must be excluded. This will be done, in the case of the three first, by a careful local examination. The exclusion of hæmatemesis, or bleeding from the stomach, is referred to elsewhere. It may suffice to recall that, as a rule, this is not difficult. The patient can frequently—not always—tell whether the blood was coughed up or vomited. It has to be borne in mind, however, that confusion may occur through the aspiration into the respiratory passages of blood coming with a rush from the stomach or other parts, and conversely through the patient swallowing blood which has come with a rush from the respiratory passages. Careful attention to the history and concomitant phenomena will usually suffice to let the distinction be made. The characters of the blood in hæmoptysis—generally more or less frothy, whether bright or dark in colour, alkaline in reaction, without microscopic appearances suggestive of stomach origin—are in many cases conclusive. In most cases of hæmoptysis, some hæmorrhagic expectoration continues for days, or at least hours. There must also be excluded other varieties of spurious hæmoptysis, such as occur from time to time in hysterical patients and malingerers.

The determination of the seat and cause of the bleeding may be easy. On the other hand, caution must be exercised in endeavouring to fix these by examination of the patient. In many cases it is sufficient to diagnose hæmoptysis, with a probability in favour of one or other cause from the history and appearance of the patient. The full physical examination of the thorax will be safely delayed. When this is undertaken, crepitations may be revealed on auscultation over an apex or other part, and other signs help to decide whether the condition is due to tuberculous disease, mitral lesion, or other of the causes which have been discussed.

Prognosis.—The lay mind tends to exaggerate the gravity of hæmoptysis. This arises, doubtless, from its relationship with the much dreaded consumption. While often anxious, the *immediate* prognosis is seldom bad. The sooner the subject of pulmonary tuberculosis realises that the incidence of hæmorrhage is an occurrence to be looked for in the course of his illness, and consequently not a cause of increased alarm, the better for himself and his physician. It is not a common event for a patient to die from such bleeding. The gravest cases are those where an aneurysm has burst from without into the respiratory passages, or where a small aneurysm of the pulmonary artery has given way. The outlook in the former instance is of the worst, and in the latter far from good, although patients recover

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from time to time. In the more frequently occurring hæmoptysis, which is less urgent, the immediate prognosis is favourable.

The ultimate prognosis will depend on the nature and stage of the disease with which the hæmorrhage is associated, and implies a careful consideration of the history and present condition of the patient. This larger prognosis must necessarily be delayed sometimes till the acute hæmorrhagic manifestations are in abeyance.

Treatment.—There should be no routine method. Every case of pulmonary hæmorrhage must be considered individually. Thus, in rupture of a thoracic aneurysm, special treatment is of little account. In other instances, as in mitral stenosis, the bleeding may be actually beneficial, and attempts to stay it correspondingly harmful. Before treatment is instituted, regard will be given, therefore, to the primary disease.

Hæmoptysis, as occurring in pulmonary tuberculosis, may be taken as a type. Here the bleeding may be fulminant in degree, as from a small aneurysm, severe as from an eroded vessel, or slighter in amount, as from congested vessels. In the slighter cases, little or no treatment is necessary. Given, however, a pronounced hæmorrhage, the first duty of the physician is to reassure the patient and the patient's friends, and so calm the patient's circulation. This is the first step towards the institution of absolute rest, which is the sheet-anchor of successful treatment. The patient should be placed in the recumbent posture, with the head raised. Perfect quiet is to be enjoined, and speaking forbidden. The room should be kept strictly cool, the clothes light, the extremities warm (if necessary with hot bottles). Ice may be sucked and iced milk drunk. The external application of ice to the chest need only be considered in the graver cases. The application of ice to other parts, for example, the external genitals, has been found of advantage. The bowels may be relieved with advantage, and best by calomel, which exerts a beneficial influence on the circulation.

The further treatment will vary in different cases. When there is much excitement of the circulation, depressant remedies will be found of value. Thus, occasionally, minim doses of tincture of aconite, which has been shown to have a powerful influence on the pulmonary circuit, may be of use, or corresponding doses of ipecacuanha. The latter drug has been largely used by French clinicians, on the authority of Trousseau, in emetic doses, say 20 grs., repeated if necessary. The results obtained in some instances are remarkable. Calomel, as already suggested, is of service in the same direction. In rare instances, it has been found of advantage to practise venesection. Sedative remedies are also of efficacy, and of these the most serviceable is opium. Except in very profuse hæmorrhage, opium may be given freely. It calms the excited sensorium and heart, and relieves cough. Sometimes the combination of opium and digitalis acts like a charm, even although the significance of the latter drug has been questioned because of its assumed tendency to raise the pulmonary pressure. The addition of a small quantity of calomel to the opium neutralises its constipating tendency beneficially.

The use of hæmostatics and astringents is much practised. Of these, the most valuable is turpentine, with its congeners, terebene, eucalyptus. These are often given advantageously in full doses. Ergot is much in vogue, given by the mouth as the liquid extract (1–2 drm. doses), or subcutaneously, as ergotin in 2–5 grs., but its results are uncertain and often disappointing. Tincture of hamamelis (30–60 minims) is sometimes of service, as also acetate of lead, the latter advantageously combined with opium. Less trust is to be placed in gallic acid and tannin, which were formerly much used, and the dilute mineral acids, which have, however, a cooling influence, grateful to the patient. The domestic remedy of common salt may be used beneficially where other agents are not available.

Where pulmonary tuberculosis is present, the adoption of the open-air treatment is to be specially commended. It is remarkable, in my experience, how uncommon hæmoptysis is when tuberculosis is treated on this system.

For some time after the acute attack the diet should be simple and non-stimulating, and alcoholic liquors excluded. After serious hæmorrhage, the consequent anæmia may require consideration. Prophylactic measures, such as the avoidance of excitement and sudden effort and the restraining of cough, will be carefully enjoined.

PULMONARY EMPHYSEMA.

A condition of permanent over-distension of the pulmonary alveoli, with associated textural changes, which produce respiratory and circulatory embarrassment.

History.—Emphysema has been known from the early-days of medicine. Within later times the condition was the subject of special study by Laennec, with whose name it has been sometimes associated. Still more recently it has been the cause of much controversy. This will be referred to again in relation to its etiology. Suffice it to say meanwhile, that three main theories of its production have been evolved :—

First, the Atrophic or Degenerative theory, which may be associated with the name of Jackson of Boston, a pupil of Louis. The theory has been elaborated by many subsequent observers. Second, the Inspiratory theory, in one or other of its modifications, associated with the names of Laennec, Gairdner, and others. Third, the Expiratory theory, associated with the names of Mendelssohn and Jenner.

Etiology.—The emphysematous lung has lost the power of emptying itself. The chest is in the position of permanent inspiration. The patient experiences expiratory dyspnœa. With the advance of the process there is interference with the pulmonary circulation and extra strain on the right heart, which in turn produces hypertrophy—usually with dilatation, and the after-results of that condition, which are considered elsewhere.

As has been said already, much controversy has taken place with regard to the causation of emphysema. It is to be feared that the consideration of the question has consequently been sometimes one-sided. The several leading views are supported by such weighty arguments, that it may be fairly assumed that there is some measure of truth in each.

Two leading factors must be admitted as playing a part in its production—First, diminution in the resisting power of the pulmonary tissues: and second, increase in the intra-alveolar tension from one or other cause. For the sake of clearness, the two factors will be considered separately.

Diminution in the resisting power of the pulmonary tissue. Emphysema is certainly more frequent in damaged lungs. It follows in the wake of bronchitis and interstitial inflammations, which presumably weaken the elasticity of the organ. It is frequently associated with atheroma and other circulatory disturbance, whereby the lung's nutrition is impaired. Comparatively seldom seen in children, it becomes growingly common with advancing age. Indeed, some degree of emphysema may be regarded as constant in the atrophic lung of the old man. The suggestion has been made that the diminished resisting power is due to congenital defect—possibly an insufficiency of the elastic tissue elements—and thus heredity is made to play a part. The occurrence of emphysema in quite young children has been cited in support of this conception. Attention has also been drawn to the early ossification of intercostal cartilages which occurs in some subjects, and the associated enlargement of the upper portion of the chest as a cause of emphysematous dilatation, the lungs distending to occupy the extra space afforded by the thoracic alteration. But the collective evidence leads to the conclusion that heredity exerts a subsidiary influence as compared with that exerted by catarrhal or other affections of bronchi and lungs, or by interference with their vascular supply. We have only to recall the extremely delicate structure of the alveolus, infundibulum, and bronchiole, to realise how disturbance—organic and functional—is effected by such processes.

Increase in the intra-alveolar tension from one or other cause. *A priori* it might be supposed that a large increase in intra-alveolar tension might suffice to produce emphysema, apart from other influence. Practically, however, this event does not occur. In the large majority of cases there is evidence of the co-operation of the first group of factors which has been discussed. Such increase in intra-alveolar tension may take place theoretically, either during inspiration or during expiration, and both possibilities have had the strenuous support of different observers. All things considered, the expiratory influence must be admitted the greater. Cough is the chief agent, which produces increase of the distending force. With the glottis closed, and the extraordinary muscles of expiration brought into play, the lungs are powerfully compressed, and tend to yield where they are least supported, more particularly at the apices, between the ribs, along the anterior edges and the bases; the left base (unsupported by the liver) being more affected than the right. The influence of forced expiration is further illustrated by the fact that emphysema tends to occur in players on wind instruments and public singers. Similarly, it is induced by sustained effort, with closed glottis, as for example in lifting heavy weights. This explains its occurrence in smiths, carpenters, and the like. It is further much more frequent in men than in women, doubtless for the same kind of reason. Such influences will be the more efficient, when the resisting power of the pulmonary tissue has been prejudiced by precedent disease.

The theory that the distension occurs as the result of inspiratory effort assumes the existence of obstructive bronchitis or other effective lesion in an area of lung. A portion of the alveolar surface being rendered useless, the inspiratory force is expended more strongly on the rest. The theory readily explains the temporary distension that occurs in one portion or one side, when another is *hors de combat*, but is less applicable in true vesicular emphysema.

Various subdivisions of emphysema have been suggested. Some of these are of little service. It is essential to separate between Temporary Distension without textural change, such as occurs in a lung when there is a large pleural effusion on the other side, and Permanent Emphysema. It is convenient to separate Partial or Local from Complete or General Emphysema, and Interlobular from Vesicular Emphysema. To some of these further reference is made under Morbid Anatomy. The term Compensatory Emphysema has been applied to the partial emphysema which occurs in relation to consolidation of other portions. The name is misleading, inasmuch as persistent cough has probably been the cause, and the emphysematous area affords no functional compensation. If a distinguishing term be needed, Complementary would be better than Compensatory.

Morbid anatomy.—**Vesicular emphysema** may be general (diffuse) or partial (limited, local). In the former—the condition which will be described clinically with greatest fulness—the lungs are large, and, when the thorax is opened, do not collapse so much as normally. They are distended, especially at the apices, anterior edges, and borders of the base. Large bullæ may project from the surface. The distended portions are of greyish or grey-pink colour, while the rest may be red. On being squeezed, they yield like a pillow filled with down (Laennec). On section, the distended bronchioles and alveoli may be recognised. On microscopic examination, the infundibula and alveoli are found enlarged, and the capillaries fewer. The interalveolar wall may have given, affording free communication between adjacent alveoli. The epithelial lining may seem intact, but the cells often show fatty degeneration. The heart is enlarged, more particularly the right side—both right ventricle and auricle distended—and the vessels are frequently atheromatous. In senile (atrophic) emphysema, there are similar changes, but the lungs are small, through advancing atrophy. In partial emphysema there is a like condition, limited in distribution, with adjacent areas of consolidation, frequently tuberculous. True emphysema must be separated from simple distension of the lung, which may occur acutely, for example, when the other lung is collapsed. In such distension there is no determinable histological change.

Interlobular emphysema.—This may be present along with vesicular emphysema, or be independent. The walls of the alveoli have ruptured from one or other cause, with escape of air into the interstitial structures. Hence the condition is sometimes known as interstitial emphysema.

Symptoms.—For descriptive purposes, the type is to be found in the subject of *Chronic General Emphysema.*

The patient is most frequently a man in middle life or beyond it, probably with a history of repeated catarrhal attacks, and perhaps of other respiratory strain. He complains especially of dyspnœa, which may be constant, and is increased on exertion. Walking up an incline, or making a sustained effort with closed glottis, accentuates it. If the condition be more pronounced, much slighter movements disturb him. He has difficulty in rising from the sitting posture. Speaking is so trying that he acquires a characteristically slow, interrupted manner of conversation. The dyspnœa is intensified during meal times. The condition may be so aggravated as to amount to orthopnœa. The patient tells of great liability to take "cold," with resulting cough and a further accession of breathlessness. Cough and expectoration are the expression of bronchitis or other catarrh, but are related causally to the emphysematous process in the manner already described.

The patient's face is livid, more or less cyanosed, and perhaps the surface radicles injected. His figure is remarkable, and much may be determined before the chest is exposed. His neck is short and often thin, with conspicuous projection of the sterno-mastoid, scaleni, and other auxiliary muscles of inspiration. The hypertrophied muscles sometimes make the neck seem thick. The jugular veins are distended, and tend to become engorged during forced expiration and coughing. His arteries are frequently atheromatous. The general form of the thorax, which we shall discuss more minutely later, is rounded, with marked antero-posterior increase. The patient may seem slightly hump-backed, from the kyphotic projection of the spinal column. His posture tends to be stooping.

Physical examination. — In addition to what has been mentioned, there is much that is characteristic. The chest is conspicuously increased in more than one direction, and in varying degrees according to the stage of the disease. There is especially an antero-posterior increase, the sternum being projected forwards, and the vertebral column backwards. The kyphosis extends from the lower cervical region downwards, but is accentuated in the lower dorsal and upper lumbar vertebræ. The transverse diameter may also be increased. The diaphragm is depressed. In most complete degree, the chest assumes the "barrel" shape. The supra- and infraclavicular fossæ tend to be less conspicuous than usual, though the thinness of the patient may allow them to be defined. On coughing there is usually some degree of bulging of the lungs into the supraclavicular areas. The direction of the ribs is more horizontal than normally. The intercostal spaces are marked, and tend to bulge on expiration and coughing. These changes begin at the upper portion of the chest, and, even when the barrel form is attained, remain more marked in the upper two-thirds of the thorax. Epigastric pulsation is a characteristic sign, due to the depressed position and the enlargement of the right ventricle. The apex impulse may not be determinable, owing to the overlapping lung. The liver may be visibly lowered. The abdominal muscles are conspicuous, especially during expiration. The respiratory excursion is limited. The chest is elevated or drawn up *en masse* rather than expanded. The respiratory capacity, as estimated by the spirometer, is reduced. Inspiration is feeble, and expiration much prolonged. During the former, the abdominal wall tends to be indrawn rather than projected. Vocal fremitus is diminished.

Percussion shows that the limits of the lung are extended both above and below, and encroach unduly on those of other organs, notably the heart and liver. The percussion sound is hyper-resonant, hardly tympanitic. The heart is mapped out with difficulty, and the outline must be regarded as approximate only. The right heart can usually be determined as enlarged. Along with the diaphragm, the heart, liver, and abdominal contents are depressed.

Auscultation reveals feeble breathing, which, when there is accompanying catarrh, is markedly harsh. Expiration is much prolonged. Fine crackling accompaniments are reported sometimes, and these have been supposed by some to be characteristic of the interlobular variety. This is very doubtful. Frequently no accompaniments are audible, in the absence of complicating catarrh, or œdema or tuberculosis. The vocal resonance is generally reduced. The heart sounds seem more distant, save over the pulmonary area, where the second sound is commonly accentuated. The dyspnœa from which the patient suffers is due to several causes. The chest is fixed approximately in the position of inspiration. The range of excursion is remarkably diminished. That means that the introduction of oxygen and the discharge of carbonic acid are correspondingly lessened. The diminished respiratory movement removes an important accessory factor from the general circulation. Further, the disruption of capillaries, effected in relation to the distended alveoli, reduces the area of interchange between atmospheric air and blood, and both directly and indirectly increases the work of the heart, the right ventricle more immediately. To cope with this, hypertrophy must be effected. As a rule, hypertrophy is accompanied by some degree of dilatation.

The progress of the patient depends on the possibility of maintaining compensation, and this on the state of the heart muscle and the auxiliary muscles of respiration, and largely on the circumstances of the patient, in respect of the possibility of sparing himself from effort, and protecting himself from catarrhal and other complications. If he can give up work, and remove himself to the protective influence of a warmer climate during winter, the evil is minimised. Otherwise the process tends to be progressive. Continued effort and recurrent attacks of bronchitis play havoc with compensation, and each extra-exertion renders him dyspnœic. He has no reserve capital, he is broken-winded. Dilatation of the right ventricle becomes more and more in excess of hypertrophy. This may lead to relative incompetence of the tricuspid valve, with further engorgement and pulsation of jugular veins, general venous stasis, and the dropsical manifestations which succeed to cardiac failure. Death results from asystole, or from the accession of a catarrhal or other acute affection, for which the patient is ill prepared. Fatal hæmorrhage has been reported, but is certainly rare in uncomplicated cases.

Interlobular emphysema.—There is little to detain us from the clinical point of view, in addition to what has been said. This variety results from the rupture of air vesicles from whatever cause effected.

Senile or atrophic emphysema.—This is a manifestation of old age. The small atrophic lung fails to resist the strain of coughing, in presence of a slight bronchial catarrh, and gives accordingly. The actual clinical manifestations are insignificant.

Partial or local emphysema.—Abutting on patches of tuberculous consolidation it is common to find areas of hyper-resonance—limited emphysema—which has been determined doubtless by atrophic changes, and the strain of coughing. Apart from their significance in the diagnosis of tuberculosis—localised hyper-resonance should put the physician on his guard—there is little to be noted.

Diagnosis and prognosis.—This is usually easy. Inspection is often sufficient. When taken along with percussion, there should be no mistake. It is important to bear in mind that emphysema may mask other conditions. Thus, under cover of the emphysematous lung, there may be lurking an aneurysm of the aorta. It is well to exclude this, more particularly in cases where there is other evidence of atheroma of the vessels. It may mask tuberculous or other consolidation. A localised hyper-resonance is always to be looked on with suspicion in this respect. Pneumothorax and heart disease, causing dyspnœa, must be excluded. Emphysema is a chronic but progressive affection. The tendency is on the whole downwards. Much depends on the circumstances of the patient, more particularly on the extent to which he can save himself from strain and protect himself from recurrent catarrhs, which are pre-eminently harmful. The immediate prognosis depends especially on the state of the circulation. When dropsy supervenes, it becomes correspondingly grave.

Treatment.—**Prophylactic.**—Two aspects of prophylaxis present themselves. First, we must guard patients, with recurrent bronchial catarrh, or other chronic respiratory disease, from the tendency to over-distension of the alveoli. This means thorough treatment of each attack, and, more particularly, the restraint of cough. There is far too much coughing in respiratory disease. This is often the result of ignorance on the part of the patient, who thinks that by coughing he aids the cure. Much may be done by explaining to the patient that the repression of every avoidable cough is a benefit done to himself. Mere irritability of mucous surfaces should be soothed by mild anodynes. The effort of coughing may be lightened by the judicious selection of expectorants, according to the stage of the condition.

Secondly, we must protect the subject of developing emphysema from those causes which lead to bronchitis. If his emphysema is not curable, it is at least possible to stay the progress of the disease. Such a patient should wear woollen clothing, and avoid close, overheated, and dusty atmospheres and sudden alterations of temperature. When circumstances admit, it is frequently extremely beneficial to remove him from a cold, moist climate to a comparatively dry, warm, and sunny region. Either the northern or the southern shores of the Mediterranean afford the kind of protective influence which is wanted, during the winter season; and minor degrees of the same beneficial influence may be obtained nearer home. Such patients often do well by a long sea voyage through sunny seas. Relief must also be given from other expiratory strain, as, for example, prolonged public speaking, singing, playing of wind instruments, and all severe strain involving forced effort with closed glottis. Hence it may be necessary to recommend a change of occupation.

Therapeutic.—When a lung is temporarily over-distended, the undue distension yields with the removal of the cause, as, for example, a pleural effusion on the opposite side. It is another question when truly emphysematous distension has to be dealt with. Here the condition is associated with textural change, and it is extremely doubtful if this can be materially affected by treatment. On the other hand, the further development may be retarded, and palliation of the more distressing symptoms may be attained. Towards this end, hygienic measures should be first considered. The diet should be sufficient but light. More particularly, it is desirable that the evening meal be simple. In adipose subjects, benefit often follows a systematic course of reduction. The bowels should be carefully looked to. Fæcal accumulation and flatulent distension must be avoided, on account of the mechanical inconvenience they induce. The functions of kidney and skin should be maintained in good order.

Of drugs, the most generally serviceable are iodide of potassium and arsenic. The two are frequently given for a prolonged period, say three or four weeks, followed by a break of a week, and then renewed. Various tonics are helpful in different cases. Thus strychnine, iron, and, especially in thin subjects or cases complicated by tuberculosis, cod-liver oil. If there be manifestations of a diathetic tendency, for example, gout or rheumatism, advantage will be gained by the treatment of this. Where dyspnœa is especially urgent, apart from any traceable cause, such as bronchitis or heart failure, opium may be exhibited with benefit. Complicating bronchitis or cardiac failure will be treated according to the lines laid down elsewhere. Great engorgement of the venous system is best met by blood-letting, the results of which are sometimes remarkable.

Pneumo-therapeutic measures are practised with advantage in suitable cases. With variation of apparatus and procedure, two methods have been followed—(1) Inspiration of compressed air. This may be effected either by the patient inspiring compressed air in a hermetically closed chamber, in which ease the increase of pressure within the chest will be balanced by the increased pressure on the thoracic wall, or by the patient inspiring compressed air through a tube, while his body is exposed to the ordinary pressure. Or the patient may inhale oxygen. (2) Expiration into a rarefied atmosphere. The two methods have been combined in various arrangements of apparatus, and may be conveniently carried out with Waldenburg's or Dupont's. In addition to the patient's expiring into a rarefied atmosphere, expiration may be further helped through compression of the thorax externally by means of an assistant. This last measure may be conveniently pursued as an aid to expiration apart from the others.

Under pneumo-therapeutic treatment many cases undoubtedly improve. It is contended that inspiration is increased in fulness—bronchial obstruction being favourably affected also—and expiration becomes more effectual. The respiratory capacity, as estimated by the spirometer, can be shown to be increased. Care must be taken that the increase of pressure and the rarefaction is duly graduated. The method should not be undertaken in presence of atheroma or important cardiac lesion, or where hæmorrhage has threatened.

PULMONARY COLLAPSE.

A condition in which the pulmonary alveoli fail to be distended, through interference with the entrance of air, either by internal or external cause, with resulting dyspnœa and constitutional disturbance.

It was only at the beginning of the present century that collapse of the lung was recognised as a condition separate from inflammatory affections. Latterly the view has been adopted that it is essentially non-inflammatory, but that it is liable to be affected by inflammatory changes.

Etiology.—It may result from obstruction to the respiratory passages. The simplest illustration of such obstruction is the presence of a foreign body. Such a body has been supposed to act in ball-valve-like fashion, so that expiration is effective while inspiration is valueless. The air within the alveoli becomes gradually exhausted, and the walls collapse. Apart from foreign bodies, such obstruction occurs in connection with inflammatory processes—simple and specific—and with new growths of the larynx, trachea, and bronchi. There is a frequent relationship between collapse and diphtheria and bronchitis, more especially in young children, in whom the respiratory capacity is primarily low. Further, in many cases there has to be reckoned the non-resistant condition of the chest wall, which yields to atmospheric pressure in presence of obstruction above. Obstruction may be caused also by pressure from without, as by a new growth. Collapse may result from the presence of pleural effusion, or pneumothorax, the lung receding in virtue of its elasticity, and finally being compressed by the advancing collection. Or it may result from weakness of respiratory muscles. This occurs frequently in connection with exhausting fevers, or through other interference with innervation. Young children—more particularly badly nourished or rachitic—are more commonly affected.

Morbid anatomy.—The collapsed lung is necessarily reduced in size. The degree to which this occurs, and the other characters presented by the lung, depend on the amount and duration of the collapse and the cause of its occurrence. The collapsed areas may be distributed irregularly throughout the lung, or the collapse may affect the base chiefly. The pleural surface is depressed over the collapsed portions, which are evidently more solid than normal lung.

In the early stage the collapsed area is generally more or less congested. Hæmorrhages are often to be discovered within the substance and on the pleural surface. The collapse of the alveolar walls necessarily interferes with the circulation. The cut surface is of dark reddish colour, smooth and glistening—suitably described as "splenified." Round about such a collapsed portion there is usually considerable œdema. Later, the tissue becomes still firmer, of bluish grey colour, the inhaled pigment and interlobular septa being conspicuously seen. At this stage the lung is spoken of as "carnified." Still later the consistence of the organ becomes tougher, "leathery," and little can be made out, save the thickened pleura and interlobular septa with interposed fibrous masses of whitish colour. This stage has been described as the "cirrhosis of collapse."

The appearance of the chest wall has been already referred to. The character of the surrounding parts will depend necessarily on the nature of the pre-existing disease.

Symptoms.—**General features.**—The affection is not a primary one, but is to be anticipated under certain conditions, to which reference has been made. The leading symptom is dyspnœa. This may supervene very rapidly or more slowly, according to the causal factor. The dyspnœa may be most distressing in degree. Associated with this is cough, which is ineffectual in overcoming the obstruction. Symptoms of more widespread constitutional disturbance supervene, notably lividity, exhaustion, prostration. The pulse is rapid and the patient restless. Death may ensue from asphyxia or exhaustion.

Physical signs.—These may not be readily separable from the signs of a precedent or concomitant condition, as for example where collapse is dependent on interference with the integrity of the pleural sac. They are more evident in cases dependent on obstruction to the air passages. In sufficiently marked cases, there may be noted inspiratory retraction of the chest wall on one or both sides, especially evident at the base, but sometimes traceable at the apex and in intercostal spaces. When the condition has become fixed there is diminution of movement. Dulness on percussion may be made out over the affected area, but may be masked by emphysema or other condition. Auscultation reveals enfeeblement or even abolition of the respiratory murmur, or vesicular may be replaced by bronchial quality. There may or may not be audible accompaniments.

Diagnosis.—The occurrence of collapse must be anticipated under certain conditions. These have been alluded to under the head of Etiology. Of special importance is the frequency of its occurrence in relation to an obstructive bronchitis, such as follows measles, whooping-cough, or other fever, more especially in children. When, in such cases, there supervene severe aggravation of existing dyspnœa, and symptoms of rapidly increasing restlessness and prostration, collapse of lung should be suspected. The diagnosis may be corroborated by inspiratory sinking of the thoracic wall. The other physical signs—less certainly determined—such as percussion dulness, bronchial breathing, or diminution of respiratory murmur, are less significant. But a careful consideration of these, along with the mode of development, will usually suffice to exclude all varieties of consolidation with which collapse of lung is likely to be confused.

Prognosis.—This depends on the antecedent disease with which the collapse is associated, and the state of the patient. Following bronchitis in the wake of whooping-cough, measles, or other acute process, the occurrence is most serious. The immediate prognosis will be guided by the amount of respiratory distress and constitutional disturbance. Marked drowsiness, convulsions, and similar evidence of nervous perturbation are unfavourable. Diarrhœa is a grave complication which prejudices the prognosis in children notably.

Treatment.—In presence of any of the conditions likely to lead to collapse, appropriate prophylactic measures must be pursued. In all cases it is of importance that the patient be treated in a large and well-ventilated room. If the obstruction be high up in the respiratory passages, as for example a diphtheritic or other affection of the larynx, tracheotomy must be considered. If the condition be one of obstructive bronchitis, our endeavour is to clear away the secretion which blocks the passages. For this purpose the less depressant emetics often prove of greatest value—as sulphate of zinc or ipecacuanha. Later, and in slighter cases, stimulating expectorants are indicated. If fluid be present in the pleural sac, it should be removed by aspiration.

When collapse has occurred, benefit will be generally obtained from a hot bath, in graver cases combined with cold douching. This may be advantageously succeeded by the free application of stimulating oils or liniments to the surface. Great attention should be given to the constitutional state. The tendency to prostration must be met by the repeated exhibition of nourishing and stimulating foods in small bulk. Along with milk or one or other form of beef-tea it is generally necessary to exhibit alcoholic stimulants. Of these the most usually serviceable is brandy. It is remarkable how rapidly and surely the patient's strength improves under such treatment. It is also frequently beneficial to administer cardiac tonics in appropriate dose, according to the age of the patient.

ATELECTASIS.

By this is meant the condition of the congenitally unexpanded lung—the permanently fœtal lung. It has been traced variously to accidents during parturition, to pressure on the bronchi by enlargement of glands, or to pressure from below upwards by abdominal disturbance. The anatomical appearances are similar to those described already. The young patient is conspicuously feeble. Dyspnœa is a leading feature, and interferes largely with sucking. The child is growingly livid. There is undue drowsiness, which is apt to be interrupted by less or greater convulsive movements. The temperature is reduced. Death follows quickly in most cases, but may be delayed for weeks or months. The prognosis, which is essentially unfavourable, will be determined by the degree of lung involvement and the strength of the patient. In approaching the treatment, the state of the passages must be looked to, and if there is obstruction it must be got rid of. For the condition itself, hot baths with cold douching will prove helpful. Sometimes artificial respiration is effectual. The surface of the body must be kept warm.

PULMONARY GANGRENE.

Gangrene of the lung consists in the death and putrid disintegration of the lung tissue. The process may be circumscribed, that is, limited in distribution to a more or less defined portion of the organ, or diffuse throughout a lobe or lung.

The condition has not been clearly separated from certain suppurative processes, or even abscess formation within the lung. We owe the description of gangrene as a distinct clinical entity to Laennec.

Etiology.—Pulmonary gangrene is not a disease of frequent occurrence. The marvel is that it is not much commoner. It is essentially dependent on successful invasion of the lung tissue by certain putrefactive organisms—probably the *Staphylococcus aureus* and *albus*. The avenue of entrance is always open and the organisms are abundant. It would appear that in health the respiratory passages have the power of destroying these, or at least of preventing successful invasion. Under certain altered conditions the tissues become less resistant, successful inoculation is effected, and gangrene results. Hence it occurs especially in enfeebled subjects, alcoholic and insane persons, and diabetics. Under such devitalising conditions, gangrene may supervene without other evident exciting cause.

But a definitely determinant cause is often traceable. This may be—the entrance of foodstuffs or other foreign body into the respiratory passages; the presence of certain lung lesions; the extension of a septic lesion of neighbouring parts by continuity, or of distant parts by embolism.

The entrance of foodstuffs or other foreign body into the respiratory passages may take place in feeble subjects, apart from disease. Or it may occur in relation to certain nerve lesions, leading to interference with swallowing, *e.g.* bulbar paralysis, dementia, and late on in a variety of diseases associated with diminished sensibility of the laryngeal mucous membrane. Along with the food, organisms obtain entrance and secure a fitting nidus, either in the decomposing foodstuffs or directly in the irritated lung. The result is a so-called aspiration-pneumonia, which in the great majority of instances becomes gangrenous. Similarly, it may result from the aspiration of unhealthy discharges, following operation about the mouth and throat, or proceeding from malignant or septic affections of the buccal cavity, fauces, larynx, trachea, pharynx, œsophagus, or even stomach, the material being first vomited and in turn aspirated. Occasionally the starting-point has been the accidental introduction of a foreign body, such as a portion of bone, in a perfectly healthy subject.

The presence of certain lung lesions.—Gangrene succeeds to precedent lung lesion comparatively seldom. It is difficult to say why it supervenes in one case and not in the next. Of pulmonary lesions which may become associated with it the most important are croupous pneumonia, bronchiectasis, fœtid bronchitis, and more rarely tuberculosis. Suppuration of hydatid cysts has similarly preceded gangrene. Pulmonary embolism may lead to it through the carriage of infected material from a distance (*v.i.*). In a small number of instances, gangrene has resulted from pressure, *e.g.* of a tumour or aneurysm, or from traumatic disturbance of the thorax.

The extension of a septic lesion from elsewhere. Malignant or septic affections of the pleura, mediastinum, œsophagus, stomach, or vertebræ may lead, by continuous extension, to secondary involvement of the lung, with gangrenous development. Or distant processes of the same nature, *e.g.* septic puerperal infections, bedsores, bone disease, may light up a gangrenous pneumonia by means of embolism.

Morbid anatomy.—As already stated, two varieties are described —(1) *circumscribed*, *i.e.* localised to a more or less definite area of the lung, as typically seen sometimes in relation to embolism; (2) diffuse, *i.e.* affecting a whole lobe or lung, more or less. These two varieties are not sharply separated from each other. Indeed, the boundary between gangrene and pyæmic abscess is not perfectly defined.

The appearances vary necessarily with the age of the gangrene. The recent gangrenous patch is of a dirty brownish black colour. This may be recognised through the pleura, which is congested, or in some cases perforated. The stinking odour, already described clinically, is conspicuous. When the affected area is cut into, it is found to be soft and shreddy. From the section there oozes forth a dirty sanious fluid, of greyish or green-black colour. The walls of the cavity are very irregular, the more resistant fibrous elements being preserved in part as bands or tags. The neighbouring parenchyma is congested, œdematous, and frequently exhibits evidence of hæmorrhage. There may be evidence of more diffuse suppuration.

In most cases the process advances by continuity or by aspiration, and the lesion becomes more general. In a certain number, perforation of the pleura may be found, and pneumothorax. In a limited number a cicatricial process may be traced.

Symptoms.—The onset of gangrene is various. In some cases it supervenes acutely, while in others the progress is slow and insidious. In the latter it may be impossible for days or longer to determine its presence. The patient may have been the subject of precedent respiratory disease, such as pneumonia, bronchiectasis, or fœtid bronchitis. Or he may be in low condition from acute illness, alcoholism, or some nervous affection. The constitutional disturbance becomes more marked. Symptoms suggestive of septicæmia supervene, or the patient sinks into a typhoidal state, with low fever of irregularly intermittent type. There may indeed be little fever. The circulation gets rapidly depressed.

More characteristic, however, is the advent of an evil odour in the patient's breath, which becomes intensified during coughing or other forced expiration. The stinking odour is still more conspicuous in the expectoration, and has been variously described as of rotten apples or of fæces. In rare cases, when the gangrene is strictly limited, no obvious fœtor may be detected during life. In some instances an offensive discharge occurs from the bowel too. The stinking expectoration is generally considerable in quantity. Like that of bronchiectasis, it tends, on standing in a test glass, to become arranged in three layers—(1) Lowest layer, compact, greenish yellow, or brown, pus like. It is here that the worst of the smell resides. Crystals of leucin, tyrosin, fatty acids, palmitin, stearin, and a variety of organisms and amorphous débris are determinable. In addition, in most cases, a considerable quantity of pulmonary elastic tissue is found, and from the expectoration there has been separated a ferment similar in action to trypsin. It is to the activity of this ferment, doubtless, that is due the solution of the softer elements of the lung parenchyma. (2) Middle layer, translucent and of less pronounced colour, consisting of serum, with considerable admixture of mucus. (3) Uppermost layer, frothy, and more or less muddy.

Some amount of hæmorrhage is frequent, and evidence of this occurs from time to time in the sputum. The patient may die from sudden hæmorrhage, due to the invasion of a vessel of sufficient size. The tendency to hæmorrhage is encouraged by the presence of a disturbing cough. Sometimes there is a good deal of pleural pain, and occasionally a superficially lying gangrenous patch may lead, during coughing or otherwise, to perforation of the pleura, with the institution of a pneumothorax or a gangrenous pleurisy, either of which may hasten the patient's end. In other cases, the patient seems to die of exhaustion, the expression of toxæmia. From time to time a case presents itself, in which death is determined by the formation of secondary abscesses within the cerebrum.

The physical signs may not be significant. In a number of instances they are not separable from those of a pre-existent lung condition. They include especially the signs of consolidation, replaced rapidly in part by those of vomica formation, possibly in an unusual situation for excavation.

Diagnosis and prognosis.—This may be aided a good deal by the history of the patient, taken along with his present condition, both general and pulmonary. Given certain precedent conditions, which have been considered above, gangrene is diagnosed essentially by the presence of the stinking breath and expectoration, with the characters which have been detailed. Corroborative evidence is obtainable by a determination of the physical signs of a rapidly developing vomica with surrounding consolidation, perhaps in an unusual situation. The presence of elastic tissue in the expectoration is in favour of gangrene, as opposed to fœtid bronchitis and most other conditions, with which it may be confused. The prognosis must depend necessarily on the conditions which have led up to gangrene, local and general, and the habits and general state of the patient. The disease is both commoner and more fatal among alcoholic subjects and persons in impaired health. The prognosis, which is always grave, is less hopeless in apparently primary cases, which are circumscribed. The diffuse variety is correspondingly more hopeless. The tendency is to go from bad to worse.

Treatment—**Prophylactic.**—The condition must be anticipated. Special care must be exercised in respect of cleanliness of mouth and throat in enfeebled, bedridden subjects, more particularly when any difficulty in swallowing exists, or the sensibility of the pharyngeal and laryngeal mucous surfaces is reduced. In some cases it will be advisable to make use of the stomach tube for feeding. Similar care will be observed in the presence of unhealthy lesions about the buccal cavity, fauces, and upper respiratory passages.

Remedial.—The treatment of the disease itself is less encouraging than might be desired. Closest attention must be given to the maintenance of the patient's strength by sufficiency of suitable nutrition and stimulants. The general line will be similar to that for septicæmia. Special attention must be directed to the free access of an abundant supply of fresh air. This is a difficulty in connection with the hospital treatment of such patients. The attempt should be made to modify the gangrenous process by the free exhibition of turpentine, terebene, oil of eucalyptus, carbolic acid, creasote, formaldehyde, or similar agent. These may be administered internally or by inhalation, or intratracheal or intrapulmonary injection. A convenient oronasal inhaler may be constructed out of pliable wire and fine gauze, which is readily saturated with the therapeutic agent. The advantage of such an inhaler is that it is light, readily applied and kept in order, and offers little obstruction to respiration. The agent may be conveniently injected intratracheally, diluted with olive- or castor-oil. The procedure is extremely simple, and the results are sometimes satisfactory. The antiseptic agent has also been injected into the lung directly. Surgical interference by incision and drainage has been attempted, but not with much success. It would seem reasonable to expect that in the near future something will be attained by means of serum therapy.

For the rest, the treatment is symptomatic, in respect of cough, pain, pyrexia, and gastro-intestinal disturbance. The complications which have been referred to must be anticipated and, when possible, averted.

PULMONARY ABSCESS.

Syn., Pulmonary Suppuration.

A localised suppuration of the lung. As stated in relation to gangrene, it is not easy to define exactly the limits of the suppurative and the gangrenous process. An acute suppurative interstitial pneumonia has been described. It might be better to drop the term abscess and speak rather of pulmonary suppuration.

Etiology.—The presence of pus within the lung may be due to a variety of causes. It is convenient to separate them broadly as follows:—

Internal.—The pus may be determined within the lung. This may supervene in relation to—(*a*) Various inflammatory affections, *e.g.* the so-called aspiration pneumonia, croupous pneumonia, pulmonary gangrene, and tuberculosis. It must be borne in mind, however, that termination of an ordinary croupous pneumonia, or pulmonary tuberculosis in abscess formation, is a rare event. (*b*) Cancerous processes of lung or respiratory passages, or suppurative lesions of the passages. (*c*) Pyæmia, through the carriage of infective emboli to the pulmonary circulation. The pulmonary embolus, laden with pus-forming organisms, becomes the focus of secondary metastatic abscess formation. Such abscesses are usually multiple and of smaller size.

External.—The pus may result from the eruption into the lung of abscesses formed primarily elsewhere, as for example in relation to the mediastinum, vertebral column, liver, or other abdominal organ—the writer has seen such eruption follow appendicitis. While theoretically this is distinct from the preceding group, and strictly speaking should not be termed pulmonary abscess, it is found convenient to review them together. During life it is often impossible to determine certainly the primary source of the pus. The only evidence available clinically may be that pus is discharged from the lungs.

Morbid anatomy.—The appearances presented by pulmonary suppuration are referred to under the diseases in connection with which they occur. The abscess which follows a local lung process, *e.g.* pneumonia, is single, or more rarely multiple, and varies in size from a cherry to an apple. It may involve an entire lobe and tend to extend outwards from the deeper parts towards the pleural aspect of the lung. The walls of the abscess cavity are irregular, and the contained space is intersected by bands of surviving pulmonary tissue. In rare instances there may be evidence of cicatrisation, with associated cirrhosis of lung. Embolic abscesses are usually multiple and of smaller size. At first apparently solid, they liquefy rapidly. When the abscess reaches towards the pleura, the pleural surface generally shows some fibrinous exudation. Occasionally the abscess bursts into the pleural sac.

Symptoms.—These are frequently obscure. The condition may not be recognised during life. One of the most conspicuous symptoms is sudden increase and change in the character of the expectoration. It assumes a more evidently purulent appearance, perhaps of dusky red tinge from admixture of blood. The smell, which at first may not be remarkable, tends to become offensive. The odour is not, however, generally so unpleasant as is the case with bronchieetatic or gangrenous discharge. Portions of lung tissue may be recognisable in the expectoration by the naked eye. Failing this, microscopic examination usually reveals the presence of elastic fibre and other pulmonary elements.

Constitutional symptoms suggestive of suppuration may be present. Pyrexia of irregularly intermittent type may show itself at first, to be replaced commonly by a return to the approximately normal curve, when free discharge of pus takes place. The circulation is similarly perturbed. These symptoms are of greater import when they develop in a patient with a history of precedent lung mischief, or with an unhealthy wound about the mouth or throat. In some instances, however, the eruption of pus occurs suddenly, without definite precedent symptoms. In such case, it is not always easy to determine whether the discharge proceeds primarily from the lung or from an empyema, or extra-pulmonary abscess which has made its way into the lung. Apart from discharge by way of the mouth, it must be borne in mind that the clinical picture may be complicated by the bursting of the purulent collection into the pleura or pericardium, or externally.

The physical signs may not be characteristic. Those of a vomica, developing rapidly in conjunction with sudden increase in expectoration, afford strong corroboration of abscess formation. Embolic abscesses, which are frequently small and multiple, may give rise to no evident phenomena.

Diagnosis and prognosis.—In many cases the diagnosis presents little difficulty. Of special significance is the comparatively sudden determination of an abundant purulent expectoration, which tends to become offensive and contains remnants of lung disintegration, more especially elastic tissue, along with physical signs pointing to vomica formation. The history of the patient, together with evidence of aggravated constitutional disturbance, may afford important corroboration. An endeavour must be made—and this will be done best by a consideration of the history—to separate between abscess of the lung proper and the rupture into the lung of an empyema or extra-pulmonary abscess. The presence of embolic abscesses may not be determinable during life. The prognosis is always grave. A single localised abscess following pneumonia, or traceable to other terminable cause, is to be regarded more hopefully than the insidious embolic abscesses associated with pyæmia. In these latter the prognosis is unfavourable in the highest degree.

Treatment.—When the diagnosis has been certainly made, and the seat of abscess localised, surgical measures should be adopted. Incision and drainage have proved successful in a considerable number of cases. When localisation is impossible, the treatment must be largely symptomatic. The free exhibition of quinine is advantageous. In a few instances spontaneous cicatrisation would seem to have been effected.

BRONCHO-PNEUMONIA.

Syn., Catarrhal Pneumonia; Lobular Pneumonia.

An inflammatory affection of the lungs, located chiefly in the air vesicles of a number of lobules, consequent usually on extension downwards of a catarrhal inflammation of the bronchi and bronchioles, less frequently on extension laterally from the affected tubes, and characterised by symptoms referable to the local catarrh, similar to those of bronchitis, and by grave constitutional disturbance, terminating often fatally in delicate subjects, especially children and old persons.

History.—The condition was long confused with pulmonary tuberculosis. A catarrhal affection of the alveoli was regarded as the first stage of the process, which most frequently proceeded to caseation and disintegration of lung tissue. This is now recognised to be erroneous. While broncho-pneumonic manifestations are present in pulmonary tuberculosis, they are an incident in the disease, which is essentially dependent on the invasion of the tubercle bacillus. Simple broncho-pneumonia results from a variety of causes, which will be considered presently, and runs a more or less acute course, terminating in recovery or fatally, according to the severity of the attack and the strength of the patient.

Etiology.—In most instances, broncho-pneumonia is a secondary process. The conditions which precede it are various. It is important to bear in mind that it seldom succeeds a simple bronchitis in a previously healthy subject. It occurs especially in children, and particularly as a complication or sequela of certain specific affections. It occurs also in old subjects, more conspicuously those in broken health. In both these classes, it is fair to assume that disturbance of the respiratory function and diminished resistance of the tissues contribute largely to the production of the disease.

In children it is most frequently preceded by measles or whooping-cough, sometimes by both. The mortality of those conditions is traceable in greatest part to this or the closely allied bronchitis with lobular collapse. Broncho-pneumonia may similarly follow influenza, diphtheria, variola, scarlatina, and other of the specific fevers, in which a tendency to bronchial complication is recognised. For the same reason it may supervene on tracheotomy. The presence of rickets, or other constitutional weakness, plays an important part in its causation. The disease is commoner among the children of the poor, where overcrowding and consequent bad air must be held as partly responsible. Even among the more educated and better-off classes, insufficient aeration of the nursery—the fatal fear of "cold"—is a factor of no little importance.

At other periods of life, debilitating influences of more chronic nature tend to induce it. Thus the bedridden patient, with deficient expulsive power, is apt to become the subject of a descending catarrh, which may terminate in broncho-pneumonia. Similarly, the fatal issue in not a few nervous lesions, such as progressive muscular dystrophy, and conspicuously bulbar paralysis, is often determined by broncho-pneumonia. In the last of these there is, in addition to the interference with respiratory function, considerable disability in swallowing. Foodstuffs thus obtain access to the respiratory tract, and, not easily ejected by coughing, are gradually aspirated more and more deeply into the lung. The aspiration pneumonia thus induced may come to be associated with gangrene of the lung, and the clinical and pathological picture be correspondingly complicated. Apart from foodstuffs, aspiration of various discharges may occur—as, after operations about the mouth, œsophagus, larynx, or trachea, with a similar result.

What is the actual determinant of the broncho-pneumonia? In the last group of cases it may be fairly assumed to be one or other micro-organism, introduced by aspiration in the unhealthy discharge. In respect of the broncho-pneumonia which follows such acute processes as measles or whooping-cough, there is more room for doubt. The condition may be regarded as an inflammatory extension from the upper air passages—a catarrhal complication; or it may be regarded as the direct effect of the particular morbific agent—essentially a manifestation of the infective process; or it may be the effect of other organisms, which, while comparatively innocuous to healthy tissues, are capable of damaging tissues which have been devitalised by the precedent disease. More than one variety of organism is usually to be found in the tissues. Thus the *Diplococcus pneumoniæ, Streptococcus pyogenes, Staphylococcus aureus* and *albus* have been described. Of this we may be certain, that broncho-pneumonia may be caused by a considerable variety of agents, some of which are of specific, some of simple nature. There can be no question that the condition may be induced by the inhalation of irritating particles or vapours.

The occurrence of broncho-pneumonia in previously healthy subjects is relatively rare. Conditions ushered in with the appearance of an acute bronchitis may develop phenomena and physical signs which can only be explained as those of a broncho-pneumonia. While physician in Edinburgh to the Forth Bridge workmen, the writer had the opportunity of observing a fair number of such cases. They differed from croupous pneumonia in respect of their clinical history. Their comparatively acute course, terminating generally in complete recovery, separated them from the more chronic forms of pulmonary consolidation. The only etiological factor which seemed available was the extreme exposure which the men necessarily suffered working in all weathers on quite unprotected elevations above the Forth. The cases formed a very small percentage of the large amount of bronchitis which was observed.

Morbid anatomy.—On surface examination, the lung may show little deviation from the normal. In some cases traces of pleurisy may exist, but these are far from constant. Through the pleura, affected areas may be recognised here and there by their bluish grey or purplish hue. Other portions may be emphysematous. As a whole, the organ looks fuller and may be felt to be firmer than normal, while it lacks the feeling of solidification characteristic of croupous pneumonia. On section in different directions, the inflammatory process may be traced from the bronchi and bronchioles downwards. Hence the condition has received the name broncho-pneumonia. The bronchial mucous membrane is hyperæmic and swollen, and the lumen filled with greyish yellow secretion. The affected areas are recognisable by their greyish red or greyish yellow colour in different parts of the section, but more especially towards the pleural surface of the lower portions. They are ill-defined and irregular in shape, and correspond with the pulmonary lobules, hence the name lobular pneumonia. Sometimes a considerable group of these lie closely together. Such a "confluent" lobular affection simulates a lobar distribution, and the condition has been described as pseudo-lobar pneumonia. In some sections, the relationship of affected lobules to a bronchus resembles that of bunches of grapes to the vine stem. Occasionally it would seem as if the parenchymatous affection of the lung extended laterally from the affected bronchus rather than terminally. If the affected areas be squeezed, a yellowish fluid is expressed. Accompanying these evidences of lobular inflammation, there is usually more or less collapse of alveoli.

On microscopic examination, the affected patches are seen to consist of alveolar spaces, grouped, it may be, round a centrally placed bronchiole. The alveolus contains a variety of cells, which distend its lumen. These are mostly endothelial, and of different sizes and ages. Many are in process of fatty degeneration. They are the result of desquamation from the lining membrane—hence the name catarrhal pneumonia. Along with these, but in less quantity, are found leucocytes, and sometimes a few red blood corpuscles. The latter are more frequently absent. It is this cellular agglomeration which can be expressed by squeezing the lung tissue, and, during life, is either discharged as expectoration, or, when sufficiently degenerated, more slowly absorbed.

Where suppuration has supervened, as for example after operations about the throat or mouth; or when gangrene has occurred, as in "aspiration pneumonia," the changes characteristic of these processes will be discovered. The organisms which may be found have been sufficiently referred to in the etiological chapter.

Symptoms.—Broncho-pneumonia occurs, as we have seen, most frequently in children, and is generally secondary to measles, whooping-cough, or other acute illness. More rarely it appears to be primary in origin, in rachitic or otherwise constitutionally weak infants. In the adult, also, it is wont to be a secondary manifestation, complicating various acute processes or following a pre-existing lesion of the respiratory apparatus. Its occurrence as a primary affection in previously healthy persons is rare, and merits separate consideration. It will be convenient to detail the leading features of these groups *seriatim*.

Broncho-pneumonia in children.—The symptoms are similar to those of capillary bronchitis, which almost certainly precedes it, and, indeed, from which it is separated by no very definite line. The little patient, in the course of measles or whooping-cough or other enfeebling affection, begins to manifest increasing dyspnœa. The rate of breathing ranges from 50 to 70, or more, per minute. Each inspiration is shallow and accompanied by much fruitless effort. The alæ nasi are distended, the auxiliary muscles brought into play, and there may be evident indrawing of the lower portion of the thorax. Expiration is prolonged, often grunting or sighing. The interference with aeration tells quickly on the appearance of the patient. He becomes listless, drowsy, and withal restless. His face grows pale, livid, and even cyanotic, and the extremities cold and bluish. Cough, which appears early, is disturbing and even painful. It is accompanied by little or no expectoration in many cases. If present, expectoration is muco-purulent, and not very characteristic. The cough becomes less urgent as interference with hæmatosis advances. There is always more or less pyrexia, of irregularly intermittent or remittent type, the maximum temperature reaching 102° to 104° F. The pulse is rapid, often 130 to 140 per minute, and correspondingly feeble. The enfeeblement of the beat becomes more marked with the progress of the lung affection and consequent oppression of the right heart. Anorexia, with furred tongue, and other feverish symptoms, also are present.

The duration of broncho-pneumonia is extremely variable. It tends to be protracted, lasting, even in favourable cases, some three weeks and often many more. The child loses strength and flesh quickly. Both subcutaneous tissues and muscles waste, and the condition of emaciation becomes extreme. Excoriation of skin surfaces or even bedsores may supervene. In cases proceeding to a favourable termination, the pyrexia yields gradually, thus differing from that of croupous pneumonia, and the constitutional recovery is similarly slow. Even in convalescence, relapses with pyrexial exacerbations are prone to occur.

Death may result from asphyxia comparatively early, or the little patient may expire more gradually from lack of aeration, drowsiness deepening into coma. Occasionally the end is sudden, during a fit of coughing; or, as frequently happens, death is the final stage of exhaustion. It is sometimes preceded by a hyperpyrexic elevation of temperature, or, in other cases, by a fall to subnormal.

It must be admitted that the physical signs are often of less importance than the symptoms. In other words, the separation of this condition from capillary bronchitis by physical examination is not always attainable. Fortunately this is of less moment, as the one condition frequently merges into the other, and the treatment is the same. In addition to visible changes in respect of breathing already referred to, percussion may reveal a certain tympanitic ring over some part of the lung—which is suspicious—and possibly here and there more or less evidently dull foci. These should be sought for posteriorly, low down, and towards the vertebral border. The percussion phenomena may for long be negative, but in the so-called confluent or pseudo-lobar variety, dulness may be early traceable. On auscultation, there are generally determinable crepitations in large numbers, medium in size, and, it may be, consonating, resembling those of capillary bronchitis. These are most abundant towards the bases. The respiration, which is in large part masked by these, is markedly enfeebled. Here and there, perhaps—though it is not always traceable—bronchial breathing may be audible and increase of vocal resonance.

Secondary broncho-pneumonia of adults.—Broncho-pneumonia may complicate the various acute processes in the adult, which have been mentioned under the head of etiology, or may supervene in relation to pre-existing respiratory disease, such as bronchitis or tuberculosis. In such cases, the possibility of broncho-pneumonia must be borne in mind, and the lungs, more particularly the bases, examined from time to time. Otherwise the condition may be determined as the actual cause of death, for the first time, at the autopsy. Of symptoms, the most important are increase of respiratory difficulty and pyrexia of irregularly remittent type. The duration of the condition varies from days to weeks. The physical signs may be, as in the child, hardly characteristic. There will probably be abundant, medium, perhaps consonating, crepitations, distributed especially at the bases—not always separable from those of œdema or capillary bronchitis. In more pronounced degrees of consolidation, percussion dulness, bronchial breathing, and increase of vocal resonance may be present.

Primary broncho-pneumonia of adults.—The occurrence of this, though not frequent, must be admitted. In the etiological chapter reference was made to a series of cases of the kind observed among the workers exposed to extremely trying meteorological conditions, during the building of the Forth Bridge. Cases of the same nature occur occasionally in ordinary practice. The affection bears resemblance in some of its features to acute bronchitis, in others to croupous pneumonia, and in others to certain manifestations of pulmonary tuberculosis. The condition is ushered in by cough and usually severe pain, referred by the patient to the chest wall on one side. There is no initial rigor. The expectoration resembles that of bronchitis rather than of croupous pneumonia. Dyspnœa is marked. The pyrexia, which is considerable, is irregularly remittent in type, and after lasting for days or a week or two, passes away by lysis. Accompanying the local process, there is often marked prostration, which gradually disappears with the cessation of the pyrexia. The course of the disease and character of the physical signs separate the condition from bronchitis and tuberculous consolidation especially. Impairment of percussion sound is traceable over more or less extensive areas—often in scattered foci—especially towards the base on one side. This disappears completely within two or three weeks, as a rule. Along with some degree of percussion dulness, there may be a tympanitic ring. Auscultation reveals, in addition to rhonchi which may be present bilaterally, indeterminate breathing, approximating the bronchial type, crepitations and increased vocal resonance over the area of impaired percussion, all of which pass away along with the percussion changes. In the course of two to four weeks, the physical condition of the chest has generally returned to the normal.

Complications.—Various complications may be present in relation to broncho-pneumonia. Along with bronchitis, of which it is often but an extension, there may be laryngitis, which still further impedes the respiration. Some degree of pleurisy is frequently present, which, especially in adults, may pass on to effusion. The occurrence of the latter ought, however, to put the physician on his guard in respect of possible tuberculous infection. Pulmonary tuberculosis is still sometimes described as if it were a development of catarrhal pneumonia. This is an erroneous conception. Either the acute exacerbation has brought into prominence a pre-existing tuberculosis—and this is not at all uncommon—or fresh tuberculous infection has supervened in relation to the damaged condition of the lung tissue. In a relatively small group of cases, more especially referred to etiologically as aspiration pneumonia, gangrene may supervene. Occasionally a suppurative process develops. In some instances—notably in feeble, badly-nourished children—the clinical picture is further prejudiced by the appearance of troublesome diarrhœa.

Diagnosis. — The separation of broncho-pneumonia from certain conditions with which it is frequently associated, more especially capillary bronchitis and collapse of lung, is not always easy. If anatomically delimitation is possible, clinically the allied conditions merge so frequently that less is to be gained by an attempt at separation than might be supposed. It is well to recognise the probability of concurrence. Of distinguishing points, however, the degree and course of pyrexia are important. The temperature tends to run higher in broncho-pneumonia than in capillary or other bronchitis, and, *a fortiori*, than in collapse. Hence pyrexic exacerbations, with increased rate of pulse and respiration, afford presumptive evidence of the development of the broncho-pneumonic process. This may be corroborated by an irregularly remittent course of the temperature, increase of constitutional distress, and the more definitely painful character of the cough. Physical signs may assist in the discrimination, though far from certainly. Thus, percussion dulness may be determinable, with bronchial breathing and increase of vocal fremitus and resonance, which would suffice to distinguish the condition from bronchitis; while the same phenomena, in combination with crepitations, which may be consonating, would help to diagnose it from collapse. Physical signs are, however, so inconstant, that very much stress cannot be laid on them. It is further remarkable how much may be obscured by the presence of a bronchitis with coarse rhonchal accompaniments.

What has just been said applies especially to broncho-pneumonia, as it develops in the wake of measles, whooping-cough, or other acute process. Under such conditions the practical significance of some of the points cited is diminished by the possible dependence of the aggravated constitutional disturbance and pyrexia on the precedent acute process more directly.

The distinction between broncho-pneumonia and croupous pneumonia is usually clear enough. Difficulty is likely to be experienced only in very young subjects. The acuter initiation, the earlier determination of head symptoms, the continued temperature, the characteristic expectoration (if it is obtainable), and the critical fall of temperature, are, some or all, sufficient to distinguish croupous from broncho-pneumonia.

Lastly, it is the imperative duty of the physician to make sure that the broncho-pneumonia is of simple nature. There must be no possibility that an insidious pulmonary tuberculosis be allowed to lurk under cover of the less serious diagnosis. The acute manifestation may have been the means of drawing attention to a consolidated patch in some portion of the lung, which has been there for long, and is of tuberculous nature. The exclusion of tuberculous affection is not always easy in such cases. No case may fairly be regarded as simple, if evidence of consolidation continue for any length of time after the subsidence of acute symptoms. Where possible, the expectoration should be carefully examined for the tubercle bacillus.

Prognosis.—The prognosis should always be guarded. Broncho-pneumonia is a chief cause of the high mortality of measles and whooping-cough. It is to be regarded as a serious complication of those and other acute processes. Its fatality is increased in proportion to the youth of the child. Under 12 months the mortality is extreme. The prognosis will be further guided by the constitution and previous health of the subject. The determination of head symptoms (somnolence, coma, convulsions) is a bad omen. On the other hand, it is remarkable how far down-hill patients may proceed and yet recovery be effected.

Acute primary broncho-pneumonia terminates as a rule favourably. Aspiration pneumonia, as might be expected from the tendency to gangrene, is a peculiarly fatal condition.

Treatment.—The prophylactic aspect of treatment is of first importance. This is especially true in respect of young children who may be recovering from acute illness, particularly measles or whooping-cough. As we have seen, it is the carelessness frequently exhibited in relation to these diseases which must be held responsible for the supervention of this grave complication. The temperature of the sickroom should be fixed at 65° F., care being taken that there is a sufficient supply of fresh air. The dietary should be of the simplest nature for some days—it may be longer—after acute manifestations have disappeared. Care should be exercised in respect of the cleansing of the buccal cavity and throat. Daily use of the tepid bath will be found of much service.

The actual treatment is similar to that of capillary bronchitis, with which we have seen the affection may be combined. It is not always easy to determine which of the two is present. Fortunately, the difficulty does not militate against the chances of recovery. The greatest care will be given to nursing, along the lines suggested already. The dispersion of moisture from a bronchitis kettle may prove advantageous. The patient's strength must be carefully maintained by a sufficient milk diet, often advantageously combined with eggs. In severe cases, the exhibition of egg-flip every two hours has proved of eminent service. In some instances, where milk is not tolerated, and, later on in many cases, light soups or teas may replace the milk advantageously. Of all stimulants, brandy is the best for children.

Local applications are beneficial. Of these the best is the modified pack. The chest is swathed in a soft towel wrung out of water—the temperature of the water is matter of secondary import. This is surrounded by a dry flannel. Such applications are renewed every one, two, or three hours, according to necessity. The results are often remarkable. Pain and dyspnœa tend quickly to be relieved, and the exhausted patient drops off to sleep. Similar benefits may be derived from the application of constant cold, by means of water circulating through a coil of rubber tubes, which is readily adapted to the chest. Such methods are fully better in results and cleaner than poultices. Equally effective as the poultice proper is the quilted jacket, covered by mackintosh. The value of this may be increased by the application to the skin of a stimulating liniment or oil. In cases where constitutional disturbance is considerable —and conspicuously where head symptoms are prominent—better results may be obtained from actual bathing. The bath may be begun at body heat, and gradually cooled to 70°. Cold effusions to the head increase its value.

The significance of drugs varies with the stage. At the commencement, with some degree of vascular excitement, benefit will be obtained by the exhibition of ipecacuanha, acetate of ammonium, and senega. Later on, when the more urgent circulatory excitement has passed, and the chief difficulty lies in expelling the sputum, carbonate of ammonium and spirit of chloroform will prove valuable. For the rest, the treatment must be symptomatic.

When convalescence has begun, benefit is to be looked for from hæmatinics (the various forms of iron), quinine, cod-liver oil. Best of all, the patient should be sent off to the country for some weeks—the longer the better.

PNEUMONOKONIOSIS.

A morbid condition of lungs, dependent on the presence of inhaled dust particles (*πνεύωμν* lung, and *κόνις* dust).

History.—Although it had been recognised by older pathologists that disturbance of the lungs was determinable by inhalation of dust particles, little attention was directed to the subject till the early part of the present century. Observations of much importance have since been made by many workers. The pigmentation of the coal-miner's lung, which for a time was attributed to alteration of blood pigment, came finally to be interpreted as the result of inhaled coal dust. This view was placed on a positive basis by the experiments and observations of Arnold and others. Arnold traced the progress of the dust particles through the lymph channels, and their deposition in different structures. What was true of coal dust was soon shown to hold good regarding a great variety of dusts produced in connection with different manufactures and occupations. These will be considered in detail presently. More recently a question of much importance has arisen as to the relationship between pneumonokoniosis and pulmonary tuberculosis. The general conclusion may be stated as follows: —(1) It is possible to have a lung packed with such particles, more particularly with coal dust, without a trace of tuberculous involvement. (2) The vitality of lungs tends to be lowered by the inhalation of dust particles, especially the more irritating stone and metal particles, and there is thus induced a liability to successful invasion by the tubercle bacillus.

Etiology.—The immediately unpleasant effects of prolonged exposure to dust inhalations—laryngeal, tracheal, and even bronchial irritation—are familiar to travellers on badly-laid railroads in a dry season, more particularly when the fuel supplied to the engine is of poor quality. Similarly, the greyish mucous discharge which is expectorated in the morning, after a prolonged evening in a badly-ventilated smoking-room, is well known. The difference in colour, on post-mortem examination, between the lungs of the patient born and bred in the country and those of the town-dweller—rosy red in the one case and more or less greyish red in the other—is remarkable. It is now generally admitted that the darker lung pigmentation is dependent on the presence of absorbed material, especially coal dust.

These simple illustrations point to the fact that the presence of the dust particles in the atmosphere is a menace to the integrity of the respiratory apparatus. What is a source of passing irritation and trifling morbid change when applied occasionally or well diluted, becomes of serious moment when applied more constantly and in concentrated degree. In other words, the great cause of pneumonokoniosis is dusty occupation.

Such dusty occupations are legion. Much has been done by legislation within recent years to minimise the dangers attendant on some of these. Thus the better ventilation of mines has reduced enormously the ill effects of a coal-miner's life. Much remains, however, to be done. It is convenient to separate three chief groups of workers—(1) Those exposed to the inhalation of coal or charcoal dust, soot, and the like. This group includes especially coal-miners. The particular form which the lung disturbance assumes has been therefore named anthracosis. (2) Those exposed to the inhalation of stone and similar dust, in which silica or silicate of alumina is a prevalent factor. This group includes especially stonemasons (hewers), millstone-makers, glass-cutters, potters, etc. In this case the condition is sometimes known as chalicosis or lithosis. (3) Those exposed to the inhalation of metal dust. This group includes especially workers in oxide of iron. The resulting affection has been termed siderosis.

This is far from an exact division. It happens sometimes that the same worker may be exposed to the inhalation of stone and metal dust, as in polishing and grinding. Further, there are other dusty occupations, which are insufficiently recognised by the classification, such as milling of different kinds, grain-shovelling, rag-sorting, and the manufacture of various textile fabrics. It seems better, therefore, to use the more general term pneumonokoniosis.

Dust particles have not all the same deleterious influence. Thus the softer particles of coal dust, charcoal, soot, flour, and the like may be inhaled in large quantity, and produce immediately little disturbance, save irritation of the passages, which may culminate in bronchitis—the bronchitis, in turn, if allowed to continue, leading to secondary results, such as emphysema. In course of time, by continued exposure, so much material, for example coal dust, may be absorbed as to give rise to an extraordinary alteration in the colour of the lung, and serious interference with its lymphatic circulation, passing on sometimes to necrosis and sloughing.

On the other hand, the sharp particles of stone or metal dust not only produce bronchial irritation, but lead quickly to a sclerotic change in the pulmonary parenchyma — fibrosis or cirrhosis — with perhaps bronchiectasis. Later on, this damaged lung falls an easy prey to the invasion of the tubercle bacillus. In the Edinburgh district, for example, stonemason's fibrosis, which is very common, is prone to lapse into pulmonary tuberculosis, as proved by the presence finally of the tubercle bacillus in the expectoration and the tissues post-mortem. It is of interest that the harder volcanic stone, *e.g.* granite, as noted by Hamilton, is not associated with the production of stonemason's fibrosis in the same degree as is the softer stratified stone, the dust of which is finer and lighter.

The course followed by the dust particles is of considerable interest, and has been the subject of minute observation. A considerable amount is entangled in the nostrils and upper air passages, and is cast off in the mucous or muco-purulent discharge from the nostrils, larynx, trachea, and bronchi. The particles are not absorbed directly through the lining membranes of these passages. Even in the bronchi such absorption is prevented by the continuous basement membrane. An unhealthy condition of the bronchi, however—such as is produced by repeated exposure to the irritation—renders it easy for the particles to reach further down the passages, till finally they invade the alveoli. Here they may be taken up by the epithelial cells. In large part, however, they pass between the alveolar cells, and so reach the lymphatic channels. In the lymph spaces they are seized on by the cellular elements and carried forwards in the stream. They become deposited, especially in the peribronchial tissue and round the branches of the pulmonary artery, forming nodular masses in the lymphadenoid structures of the lung. Thus bronchitis is succeeded by peribronchitis. The particles further invade the interlobular septa, the deeper layer of the pleura, and the bronchial, retrobronchial, and tracheal glands. Experimental observation has suggested that inhaled pigment particles may even pass to the general circulation and be deposited in the liver and spleen.

The result of the presence of the particles in the glands is pigmentation and hyperplasia, with associated enlargement and hardness. The result of their presence in the lungs depends on the nature of the particles. Coal dust may be inhaled in large quantity and give rise to little else save pigmentation. In still larger quantity it may lead to interference with lymphatic circulation, which in turn may cause necrosis and sloughing. On the other hand, stone dust rapidly leads to a hyperplasia of connective tissue elements and the production of a sclerosis (fibrosis, cirrhosis) of lung parenchyma. As already stated, such a damaged lung becomes not infrequently the seat of tuberculous inoculation.

Morbid anatomy.—The morbid appearances vary somewhat with the nature of the inhaled dust. It is convenient to take two types—coal-miner's lung (anthracosis); stonemason's lung (lithosis or chalicosis). In other varieties the different appearances are chiefly dependent on the colour of the inhaled dust, as for example oxide of iron, which produces a reddish colour in the affected areas.

Coal-miner's lung.—The pleural surface presents a bluish black colour, with irregularly-disposed nodulations of darker hue. Section shows the organ to be more or less densely packed with pigment. The pigment is darkest in relation to the nodular areas, which are now seen to be scattered throughout the lung parenchyma. The bronchial glands are large, firm, and similarly pigmented. Apart from the accumulation of pigment, the lung may show little change. There may be pleural adhesions. There may be evidence of emphysema, and there is generally bronchitis and œdema. There is little, if any, sclerotic change, which is so marked a feature in stonemason's lung. In advanced cases there may be evidence of necrosis and sloughing, which is, however, free from the offensive odour of gangrene. Such pronounced cases are less common than apparently they used to be.

On microscopic examination the pigment is found in the alveolar epithelium and interspaces, the tissue round the bronchi and pulmonary artery, the interlobular septa, deeper layer of pleura, lymphadenoid structures, and bronchial glands. In larger particles it has been found possible to identify the particular band of coal, and in charcoal dust the texture of wood has been made out.

Stonemason's lung.—The organ is often bulky, but may be shrunken, and feels unduly firm. This resistance is especially marked in nodular areas scattered throughout the surface and substance of the lung. The pleura frequently shows adhesions and cicatricial puckering. On section, the hard nodules cut with difficulty, being tough and gritty. The bronchial glands are still larger and harder than in coal-miner's disease. In addition, there may be evidence of bronchiectasis, and, in later stages, of tuberculous affection. Gangrenous sloughing has been noted. On microscopic examination, the nodules are found to be largely composed of fibrous elements, with interposed stone dust and probably some pigment particles. Chemical examination has proved the mineral character of the minute fragments thus embedded. When fibrosis is considerable, or the lung is emphysematous, the right heart will probably be found to be dilated.

Symptoms.—The clinical features are not very characteristic. They are in large part those of chronic bronchitis or chronic phthisis. Cough is usually troublesome, yielding at first when the patient is removed from exposure to dust inhalation, but latterly persistent. Dyspnœa gradually appears, and, later, comes to be a conspicuous symptom. Expectoration is present in varying quantity. In character it may differ little from that of bronchitis. Sometimes there is evidence of the particles inhaled, for example, cotton or other fibres, gritty masses, grains of oxide of iron, or coal dust. The sputum may be actually black, although, on the other hand, there may be enormous pigmentation of lung without a trace of it in the expectoration. In other cases, more especially in the latter stages of stonemason's phthisis, many of the features of pulmonary tuberculosis show themselves, and the tubercle bacillus may be determinable. From time to time hæmoptysis occurs.

The physical signs may be those of chronic bronchitis, probably accompanied by emphysema. Where fibrosis has developed to any extent, signs pointing to consolidation may be looked for. Or again, when bronchiectasis or sloughing or tuberculous vomica has developed, there will be signs of cavity formation, such as have been detailed elsewhere. In most cases there is evidence of dilatation of the right side of the heart.

The progress of the case is generally slow. With the continuance of the causes, the patient's condition gets gradually worse. With the advance of the process dyspnœa increases, and pallor, lividity, and general feebleness ensue. Death may be determined by gradual exhaustion, or any of the causes which lead to a fatal termination in chronic bronchitis, emphysema, gangrene, or pulmonary tuberculosis.

Diagnosis.—This is not usually difficult. The presence of lung symptoms in a patient whose occupation exposes him to dust inhalations generally warrants the diagnosis. Further examination will be necessary to determine the presence of embarrassment of the right heart, as caused by associated emphysema or the advance of fibrosis. In the later stages, especially of stonemason's lung, the expectoration should be carefully examined for the tubercle bacillus.

Prognosis.—This is dependent on individual susceptibility, and the possibility of removing the patient from the noxious influence. In course of time some patients become marvellously tolerant of the irritating particles. On the other hand, when symptoms continue, there is great danger of aggravation of the patient's condition, by the development of emphysema or the advance of fibrosis and consequent embarrassment of the pulmonary circulation, with dilatation of right heart and further circulatory failure. If sloughing or gangrene or tuberculosis supervene, the prognosis is correspondingly grave.

Treatment.—The most important aspect of treatment is prophylaxis. This is a large question, and includes both individual and public measures. For the individual it may mean change of employment. This may be complete, or consist in transference from one department to another. Thus the building mason is saved from risks which the hewing mason runs. A similar principle is applicable to other industries. In like manner, the individual must be taught to recognise the danger he runs, and the extent to which this may be modified through personal care and cleanliness. The general measures include enactments regulating, for example, the ventilation of mines and other work places. In the case of anthracosis, considerable diminution in the severity of the disease has already followed such legislation.

The therapeutic measures are similar to those recommended for chronic bronchitis, emphysema, embarrassed pulmonary circulation, bronchiectasis, and chronic pulmonary tuberculosis, and need not be specially discussed. Apart from prophylaxis, they are necessarily largely symptomatic.

CHRONIC INTERSTITIAL PNEUMONIA.

Syn., Cirrhosis; Fibrosis of Lung.

A chronic affection of the lung, represented clinically by cough, dyspnœa, expectoration of varying characters, and physical signs of shrinking, dependent anatomically on progressive increase of fibrous tissue elements at the expense of alveolar spaces, and resulting from a variety of causes. Fibrous increase of interstitial elements occurs in relation to a number of different diseases, *e.g.* pulmonary tuberculosis, pneumonokoniosis, which come up for separate review. If such recognised diseases are excluded, the number of cases of chronic interstitial pneumonia is reduced almost to the vanishing point. The affection would thus appear to be an anatomical rather than a clinical entity, with diverse etiological relationships.

History.—The condition of chronically advancing lung disease, with post-mortem evidence of increase of fibrous elements, was described under one or other of the above-mentioned names long before the certain recognition, through the discovery of the tubercle bacillus, of the clinical and etiological identity of the various forms of pulmonary tuberculosis. Without doubt, a large proportion of cases which were formerly included in the category of chronic interstitial pneumonia must now be classed in the large group of chronic pulmonary tuberculosis. Similarly, the condition was described and named before there was any general recognition of the intimate relationship which has since been traced between the inhalation of certain dust particles—especially stone dust—and the production of fibrosis of the lungs. The changes produced in the lungs by the inhalation of dust particles have been recently the subject of careful observation, and the various disturbances have been grouped together under the term pneumonokoniosis, to which a special chapter in the present work is devoted. With the advance of etiological research in lung disease, there has been a remarkable narrowing of the limits of chronic interstitial pneumonia as a clinical entity. It is a question whether the name should not be dropped altogether from the clinical nomenclature of disease.

Etiology.—Apart from certain well-recognised diseases with chronic cause, in which pulmonary fibrosis plays an important part, chronic interstitial pneumonia is of rare occurrence. It is open to doubt if it ought to be admitted as a separate disease. It will be convenient, therefore, to consider generally what are the causes which give rise to increase of pulmonary fibrous tissue. Such fibrosis or cirrhosis is determinable by a variety of exciting agents. It may result from the action of irritants conveyed to the lung, either by way of the air passages and thence by the lymphatic channels, or by way of the blood; or by extension of a pleural condition.

Of irritants giving rise to the condition, the most familiar example is inhaled dust, especially stone dust. From this point of view, a stonemason's lung is chronic interstitial pneumonia, dependent on the irritation of the silicate particles, which on inhalation pass into the lung by the interspaces between the epithelial cells of the alveoli, and so reach the lymphatic circulation. Their further cause and effects are considered in the chapter on Pneumonokoniosis. The tubercle bacillus acts likewise as a determinant of fibrosis, the fibrous tissue which results being doubtless limiting, and therefore protective. Similarly, catarrhal pneumonia, which itself is determined by some irritant — conspicuously the catarrhal pneumonia which follows measles and whooping-cough—may be prolonged into a chronic interstitial pneumonia. Very rarely, if indeed at all, is fibrosis determined by a precedent croupous pneumonia. In most cases where this may seem to have been the case, the lung was damaged prior to the attack of croupous pneumonia. Of irritants acting on the lung more directly through the blood, syphilis may be taken as an example. In the syphilitic affection of the lungs in infants—so-called white hepatisation —and the more irregularly distributed syphilis of the lungs in adults, there is considerable manifestation of fibrosis.

Fibrosis of the lung may result by extension of a pleural condition. This may be either a dry pleurisy, or a pleurisy with effusion. The pleurisy is often, however, but an early manifestation of tuberculosis. Adhesions occur, the pleural layers are thickened, and along with them the interlobular septa. This process affects especially a lower lobe, and may be confined to this, but may be more general. It is not always easy to say whether the lung process has been secondary to the pleural, or conversely. The continuity of vascular and lymphatic supply between lung and pleura explains why either occurrence is possible.

The essential condition to the production of lung fibrosis is long-continued irritation of the lung parenchyma. The irritant may be of different kinds, and may enter by different channels. In many cases the fibrosis, as for example in chronic pulmonary tuberculosis and pneumonokoniosis, is recognised as an essential part of the disease. Chronic interstitial pneumonia would thus appear a convenient term by which to describe an anatomical result, although the etiological relationships and the clinical features are widely different. It is conceivable, though by no means proved, that in addition to the groups already admitted there is a small remaining group, in which the process of fibrosis slowly advances in the lung, apart from other recognised lesion, as it advances in the kidney, liver, and other organs.

Morbid anatomy.—Sufficient has been said under the head of Etiology to permit of a reference in respect of anatomical appearances to the chapters which deal with the conditions of which chronic interstitial pneumonia forms a conspicuous part, *e.g.* pneumonokoniosis, chronic pulmonary tuberculosis, bronchiectasis. The affected portion of lung is resistant and airless. On section, strands of light or darker grey colour and of varying size are traceable. These amount sometimes to dense, almost cartilaginous bands or areas. In such ease, microscopic examination may reveal merely fibrous elements, without a trace of lung tissue. The pleura is frequently much thickened, and its layers adherent. There is commonly evidence of bronchiectasis. This may be extreme. The unaffected portions of the other lung are probably emphysematous. The right heart is enlarged, with more or less pronounced dilatation of the right ventricle.

Symptoms.—**General features.**—The affection is a chronic one, and the symptoms vary according to the stage of the disease. They do not differ much in the main from those of chronic pulmonary tuberculosis, pneumonokoniosis, or even chronic bronchitis. Cough and dyspnœa are present in less or greater degree. Expectoration is of variable quantity, muco-purulent, perhaps fœtid, and may present the other characters of bronchiectatic discharge. Absence of the tubercle bacillus is an important negative character. Hæmoptysis occurs frequently, and this may become so severe as to lead to death. In other cases the patient may live for many years, and the process be comparatively quiescent. The continuance of the condition exerts a strain on the right heart, which in turn may fail. With failure of the right heart, œdema of the extremities and viscera may supervene, and death result. For a long time the general condition of the patient may be little disturbed. The temperature may be almost constantly normal or subnormal, though elevation of temperature occurs from time to time. With the continuance of the disease, the patient tends to get feebler and thinner. The finger-nails are frequently clubbed.

Physical signs.—On inspection and palpation, there is evidence of retraction of one lung—it may be of both—with resulting deformation of thorax, both anteriorly and posteriorly. The vertebral column may participate in the deformation. Mensuration shows a reduction in the size of the affected side, and a cyrtometric tracing reveals the flattened outline. The excursion of the thorax is limited. Vocal fremitus is often increased, but may be diminished. Percussion yields dulness in part, with probably here and there tympanitic areas corresponding to the position of bronchiectatic cavities. If one lung only is affected, the other lung is frequently hyper-resonant. Auscultation reveals feebleness of respiratory murmur, perhaps of bronchial quality, often accompanied by crepitations, fine or coarse. Cavernous or even amphoric breathing may be audible over bronchiectatic cavities, along with other signs suggestive of vomicæ. Vocal resonance is generally increased, but may be diminished.

There is commonly evidence of dislocation of other organs. Thus the heart may be drawn to one or other side by the retracting lung. Similarly the diaphragm on the affected side may be elevated, and the corresponding abdominal organs (liver, stomach, spleen) displaced upwards. With the continuance of the process, the right heart becomes enlarged; and with excess of dilatation over hypertrophy corresponding evidence will be forthcoming. With failure of right heart, relative incompetence of the tricuspid valve may be established, with engorgement of veins and œdema of extremities and viscera.

Diagnosis and prognosis.—The determination that a condition of fibroid change is present is usually not difficult. There is evidence of shrinkage of one side of the thorax, or a part of one side more especially. There is generally evidence of bronchiectasis. There may be evidence of displacement of the heart and other organs, and some degree of spinal deformity. The more difficult side of diagnosis is the determination of the etiological relationships of the fibrosis. In particular, pneumonokoniosis must be separated as a distinct condition, and chronic pulmonary tuberculosis carefully excluded. If this be carried out rigidly, the number of cases of chronic interstitial pneumonia occurring in practice will be found to be small. Occasionally the therapeutic test, in view of a possible syphilitic relationship, will prove valuable. The advance of the process is generally slow. The prognosis is either that of the condition with which it is associated, or of the conditions to which it may give rise, especially the circulatory embarrassment.

Treatment.—Little can be done for the process itself. Attention will be directed prophylactically to the removal of influences, *e.g.* of occupation, which may be inimical to the lung. Consideration of the etiological aspects may suggest, in certain cases, the exhibition of constitutional remedies, as against tuberculosis or syphilis. For the rest, treatment must be largely symptomatic, and similar to that recommended for bronchitis, bronchiectasis, pulmonary tuberculosis, and other chronic lesions. Special attention should be directed to the state of the heart and circulation.

PULMONARY SYPHILIS.

Etiology.—Considerable difference of opinion has been expressed as to the frequency of syphilitic manifestations in the lung. Much depends on what is included under the description. Respiratory symptoms are common enough in patients who have been affected by syphilis previously. But it may be incorrect to assume that these are syphilitic manifestations, even although the patients seem benefitted by specific treatment. Moreover, as matter of experience, only a small number of patients with lung symptoms and signs appear favourably influenced by such treatment. The frequency of actual syphilitic lesions within the lungs proper is not very great. Commoner than pulmonary lesions are lesions of the larynx, trachea, and bronchi. These are considered elsewhere. Syphilitic lesions of the lung may result from inherited or acquired disease. In the latter case, they appear as tertiary manifestations.

Morbid anatomy.—In the adult, syphilis of the lungs takes the form generally of a patchy cirrhosis. Fibrous strands are scattered irregularly throughout the lung. They seem to occur most frequently about the root of the lungs, the middle lobe of the right lung, less frequently at the apices. They may occur also at the bases. The pleura is probably involved, and the contraction of the fibrous strands leads to a certain amount of shrinkage of the lung; or gummatous changes supervene. Over such areas the pleura is frequently thickened and puckered. There may also be evidence of syphilitic involvement of the respiratory passages, larynx, trachea, or bronchi.

In the infant, inherited syphilis may manifest itself in the form of so-called white hepatisation (pneumonia alba) of the lungs. The condition is one of pathological rather than clinical interest, and does not call for description here.

Symptoms.—The features are not very characteristic. They are similar to those of chronic pulmonary tuberculosis or new growth, with each of which there is liability of confusion. A history of syphilis may usually be obtained. There is probably evidence of syphilis elsewhere. Manifestations of syphilis in the respiratory passages, determinable by inspection, should be regarded as of special significance. The condition sometimes yields quickly to specific treatment, but its course tends to be chronic.

Diagnosis and prognosis.—In obscure chronic lung disease, the possibility of syphilis will be thought of. The fact of a syphilitic history is important. Still more so is the presence of undoubted syphilitic lesions elsewhere, especially in the respiratory passages. The therapeutic test, though far from certain, may afford corroborative evidence. Negative evidence in respect of other conditions, more especially pulmonary tuberculosis, should be carefully sought. The local condition of the lung is not likely to lead to dangerous results.

Treatment.—This is carried out on the same lines as for syphilis generally.

NEW GROWTHS.

New growths of the lung are not very common. Although forming clinically but a small fraction of pulmonary disease, the group is one of much interest and significance. It includes carcinoma, sarcoma, lymphosarcoma and enchondroma. Of these, carcinoma is the most frequent and important.

Etiology.—The tumours are either primary or secondary. Primary new growths are comparatively rare. The great majority are secondary.

Chief of the primary new growths is carcinoma. It occurs especially in adults from 40 years of age and onwards. The tumour, which is generally of considerable size, affects one lung only, as a rule. Occasionally both lungs are involved, or at least secondary manifestations are present in the other lung. The right side would seem to be more commonly implicated, preferably in its upper portion. The tumour, which is most frequently a cylindrical epithelioma, commences in the epithelium of the bronchial tubes, and spreads by continuous extension and by way of the lymphatic circulation. The pleura commonly becomes involved. With increasing bulk of the growth, pressure is exerted on the lung tissue, the respiratory and other passages, vessels, and nerves, and corresponding symptoms are induced. The bronchial glands are usually affected, and frequently the axillary and cervical. Apart from those occurring in the pulmonary structures and adjacent glands, secondary manifestations are not often found in other organs. They have been reported in the brain and liver. The further etiology of the growths is that of tumours generally, and does not call for consideration here. Reference should be made, however, to the comparatively common appearance of a variety of lympho-sarcoma among the workers in the cobalt mines of Schmeeberg in Saxony. Its frequency in this restricted area is of significance in relation to the etiology of tumours generally.

The chief members of the secondary group are the carcinoma and the sarcoma. They may occur at almost any age in presence of a primary growth elsewhere, of which they are metastatic developments. The primary growth may be situated in the mammary or other gland, gastro-intestinal tract, genito-urinary apparatus, or bone. The secondary growths result from the involvement of a vein by extension of the primary mass and the carriage of an infective embolus to the pulmonary circulation. They may also originate through carriage by way of the lymphatic channels. There may be a history of operative interference, and apparently successful extirpation of the primary tumour.

Morbid anatomy.—Little need be added to what has been said under the head of Etiology. The primary tumours include carcinoma, sarcoma, lympho-sarcoma, and enchondroma. Excluding the last, which is comparatively rare and unimportant, we may consider one or two of their leading features. In contrast with the secondary tumours, they are usually single and form one continuous mass of considerable size, ill-defined, and extending irregularly into the surrounding tissues. The growth tends to compress the lung, which gradually loses all trace of its structure, and is converted into a soft, friable, airless mass of greyish colour. The pleura may show inflammatory changes, and there is frequently evidence of effusion—commonly hæmorrhagic. Other organs are similarly compressed, with results which have been already considered. The carcinoma has its origin generally in the bronchial wall. From this it extends to the peribronchial lymphatics, and thence more widely and rapidly. The bronchial and other glands are usually involved. The sarcoma starts from the fibrous tissue about the root, and thence invades the lung tissue proper, reaching it may be to the pleura, and even projecting externally from the surface of the chest wall. The lympho-sarcoma originates in the bronchial or mediastinal glands, and thence extends to the lung. The tumour tends to assume large proportions.

The secondary tumours include all the tumours which tend to form metastases, the more commonly occurring being the carcinoma and sarcoma. They originate by embolism of an infective particle carried from the primary tumour, wherever seated, or by carriage through the lymphatic channels. In contrast with the primary tumours, they are generally multiple, roundish in form, and well defined.

Symptoms.—**General features.**—The symptoms are not always characteristic. The clinical picture may be little different from that of a chronic—or even an acute—pulmonary tuberculosis. In a patient with primary cancer elsewhere, or after operation for the removal of a cancerous mass, the advent of such lung symptoms will naturally have greater significance. In some cases the gradual advance of the cancerous cachexia is evident. In others this is not conspicuous. With increasing size of the new growth—particularly primary cancers—symptoms referable to pressure are wont to become evident.

Dyspnœa is a conspicuous feature. This may supervene gradually in relation to advancing compression of lung tissue, or more rapidly through pressure on the trachea or bronchi. Along with this a distressing sense of oppression is often remarked. Pain is present in varying degree, and may be interpreted as due to implication of the pleura, or to pressure on sensory nerves of the thoracic wall. Cough is often troublesome, and accompanied by much aggravation of pain. Expectoration may differ little from that of other chronic respiratory affections. On the other hand, microscopic examination may reveal the presence of cancerous cells. The discharge may also be characteristically blood-stained. The staining is lighter or darker in different cases—the expectoration being then aptly likened to red or black currant jelly. In some cases it is more liquid, prune-juice like. Occasionally, serious hæmorrhage occurs, which may lead immediately to the fatal issue. As the growth increases, symptoms of pressure become more conspicuous. These may be referable directly to the tumour or to glands, *e.g.* bronchial, tracheal, or even axillary and cervical. Thus veins may be pressed on, giving rise to turgescence of their peripheral distribution, and of anastomosing branches, and swelling and œdema of tissues. The head, neck, and upper extremities—perhaps on one side only—may be disturbed in this fashion. Or pressure may be exerted on important passages, *e.g.* œsophagus, trachea, bronchi, with corresponding symptoms. Or it may be on nerves, *e.g.* the brachial plexus, with resultant neuralgia and pains, or the recurrent laryngeal, with laryngeal paralysis. Or other portions of the vagus or the phrenic may be involved. Pleural effusion is not infrequent, whether the pleura be evidently involved or not. The fluid is commonly, though not certainly, hæmorrhagic in character. Whether fluid be present or not, the patient tends to lie on the affected side, but this is not a constant rule.

The course of the disease is various. A year may be stated as an average duration from the time that the diagnosis is made. But the progress may be much more rapid. Some cases, indeed, run an acute course, like acute miliary tuberculosis. In other cases, death may be delayed for a couple of years. With the progress of the disease, there is more or less evident development of the cancerous cachexia, and the patient's strength gradually ebbs away. Throughout the course, perturbations of temperature frequently occur. The cause of these irregular bouts of pyrexia is not always clear, and they may prove misleading.

Physical signs.—These, taken separately, are not characteristic. Their collective presence, however, in a difficult case will point to the lung as the seat of disease, and their unusual distribution will serve to exclude certain of the more commonly occurring pulmonary lesions. The signs are of more certain value in primary than in secondary lesions. There may be evident projection of one side of the chest with diminution of movement. The intercostal spaces may be normal or obliterated, especially where pleural effusion has occurred. Palpation corroborates inspection in respect of altered form and diminished movement, and shows vocal fremitus to be variously affected, in some instances increased, in others apparently diminished or undisturbed. Percussion reveals dulness—frequently absolute—over a greater or less area. The sense of strong resistance is often striking. On auscultation, the respiratory murmur is frequently enfeebled, though bronchial in quality. Sometimes it is hardly audible. Crepitations are often traceable, and it may be rhonchi, or coarser accompaniments in relation to pressure on the passages. Vocal resonance is variously affected in the same way as vocal fremitus. When the pleura is affected, friction may be determined, and, with advancing effusion, the physical signs of pleurisy with effusion.

Diagnosis.—This is sometimes difficult. The very difficulty of diagnosis, however, may be a point in favour of a new growth. The history of the patient may be important. The occurrence of lung symptoms in a patient with primary cancer elsewhere, or after operation for such a growth in any portion of the body, should arouse suspicion of metastatic deposits in the lungs. The presence of enlargement of axillary or cervical glands is not opposed to the diagnosis. The simultaneous advance of ill-health and emaciation, with sallow, anæmic facies, and of lung symptoms and signs, which cannot be explained along other lines, is most suggestive. All other possibilities must of course be carefully excluded, more especially chronic varieties of pulmonary tuberculosis. Unilateral disposition and unusual character of physical signs may be of importance in this connection. Sometimes the strongest corroboration is derived from the appearance of pressure symptoms, *e.g.* engorgement and œdema of head and neck of one or other upper extremity, or paralytic or neuralgic disturbance. The expectoration, while affording negative evidence in respect of tuberculosis and other conditions, may contain suspicious cells, or have the peculiar black currant jelly or prune-juice aspect already referred to. The presence of a hæmorrhagic effusion in the pleural sac is significant, and the effusion may also contain cells suggestive of a cancerous origin.

Prognosis.—This is pre-eminently unfavourable. The fatal issue may occur rapidly—the disease running a rapid course, like acute miliary tuberculosis. In the greater proportion of cases the progress is more gradual. Death ensues usually within eighteen months of the diagnosis being established.

Treatment.—Symptomatic measures are alone possible. The indications for these are similar to those in bronchial and pulmonary disease.

HYDATID DISEASE.

Etiology.—Invasion of the lung by the echinococcus is not infrequent. Out of a large series of recorded cases of hydatid affection, in fully 8 per cent. the respiratory organs were the seat of disease. In many instances, however, the invasion of the lungs is found to be secondary to that of other organs,—as, by perforation through the diaphragm from the liver or by way of the blood stream. Primary affection of the respiratory organs is not so common. The cysts may develop in the lung proper, or in the pleura. The former is the more frequent seat. In either case, if small, they may give rise to no respiratory symptoms, and the general health remain unimpaired. When larger, their presence is manifested by pressure effects or secondary inflammatory changes, perhaps suppuration or gangrene. Cysts or portions of cysts, or their contents, may be discharged in various ways, to be referred to presently. Other points in relation to the etiology, such as their geographical distribution, are discussed in another chapter.

Morbid anatomy.—Apart from what has been said above, this need not be discussed here. Further consideration of the anatomical appearances will be found in relation to hydatids elsewhere, and in the sections dealing with Suppuration and Gangrene of the Lung.

Symptoms.—There may be an absence of symptoms altogether.

On the other hand, the patient may suffer from recurrent—it may be daily—hæmoptysis, cough, pain in the chest, breathlessness, while his general condition seems little prejudiced, apart from anæmia in case of frequent hæmorrhage. Or graver symptoms, pointing to inflammatory disturbance or suppuration or gangrene, may supervene, and the patient's general condition suffer correspondingly. The physical signs are not characteristic. They may include some degree of bulging of one side, diminished movement, dulness, enfeeblement of breath sounds, diminution of vocal fremitus, and resonance. Occasionally, fluctuation and vibratory thrill may be determined. There may be displacement of other organs. When suppuration or gangrene has supervened, signs suggestive of the condition may be present. There may thus be little in the clinical features to separate the case from pulmonary tuberculosis, pleurisy, or sometimes suppuration or gangrene. Doubtless many cases of hydatid of lung have been misinterpreted, up to a certain point. But the patient may expectorate one or more hydatid cysts, or a portion of the contents of one. Or in a purulent discharge, *per vias naturales* or otherwise, hooklets or portions of membrane may be determined. The progress is very various in different cases. Sometimes a sharp hæmorrhage leads to the fatal issue. The patient is liable to a host of accidents and complications, any of which may terminate his life.

Diagnosis.—In cases with obscure pulmonary symptoms, the possibility of hydatid disease must be kept in view. More especially is this a wise precaution when hæmoptysis recurs, in the absence of other evidence of pulmonary tuberculosis or circulatory disease. Expectoration, sanguineous or other, should be carefully searched, for the presence of portions of membranes or hooklets. When, as sometimes happens, a complete cyst is discharged with a cough, there can be no further doubt. In the absence of such positive evidence, it is only possible to proceed by the careful exclusion of other conditions. Exploratory puncture may prove of value, where physical signs are sufficient to localise. It is to be borne in mind that a suppurating hydatid may be thus discovered, even in the absence of pyrexia or marked constitutional disturbance. The possibility of a primary seat in the liver, with rupture into the lung or pleural sac, must be borne in mind, as also the presence of a complicating pleurisy with effusion, which the writer has seen on more than one occasion. The history and residence of the patient, as pointing to liability to infection, will be of moment. This is referred to more in detail elsewhere, in connection with the geographical distribution of hydatids.

Prognosis.—The outlook is anxious. In some instances the patient apparently expectorates the hydatids entirely, with a disappearance of symptoms. The possibility of accident or complications is imminent. Thus the patient may die of hæmorrhage or inflammatory changes, with suppuration, or gangrene may supervene, and lead to death. Or perforation may occur into other structures, *e.g.* pleura, pericardium, peritoneum, with attendant risks. Suppuration and discharge externally may have a favourable issue.

Treatment.—So soon as the diagnosis is certain, operative measures should be considered, and, where practicable, carried out. The details of these need not be entered on here. In a certain number of cases, aspiration with a fine needle has sufficed. Short of operative interference under suitable conditions, therapeutic measures are not of special value. The treatment is in most part symptomatic. R. W. PHILIP.

DISEASES OF THE PLEURA.

PLEURISY—PLEURITIS.

INFLAMMATION of the pleura may originate under many conditions, may give rise to symptoms varying in amount and severity, and may be accompanied by anatomical changes of considerable diversity. In classifying cases of pleurisy, therefore, the differences in their mode of origin, in their clinical symptoms, or in their anatomical appearances, may be adopted as a basis. Thus as regards etiology, pleurisy is primary or secondary; as regards symptoms and course, acute or chronic, and as regards anatomical appearances, dry or adhesive, fibrinous, sero-fibrinous, or purulent, according to the amount and the nature of the exudation which accompanies the inflammation. It is convenient to employ these several methods of classification in the description of the affection.

Etiology.—**Primary pleurisy** comes on with greater or less suddenness in those who are apparently healthy, even attacking the particularly strong and robust, as an acute affection which from its mode of onset and clinical history greatly resembles an acute specific disease. In a certain proportion, but not by any means the majority of cases, it is possible to obtain a history of a chill arising from some exposure to wet or cold, contracted some hours, from twelve to forty-eight perhaps, before the onset of the symptoms of the affection.

Formerly, primary pleurisy was considered to be idiopathic in origin, but evidence seems to be accumulating to stamp the cases in which a history of a chill cannot be obtained (as indeed some in which it can) as tuberculous, as being in fact due to the presence of tubercle bacilli. The points in favour of this view may be briefly mentioned. In the first place, tubercle bacilli have actually been demonstrated in the exudation in a few cases of acute pleurisy, and in a much larger number the exudation itself when injected into suitable animals has produced tuberculosis in them. This last experiment has been successful in upwards of 60 per cent. of the cases tested. In addition to this experimental evidence, Netter has shown that many patients who have been attacked by pleurisy *a frigore*, react to injections of tuberculin almost as readily as cases of confirmed tuberculosis, and, on the other hand, Debove and Renault have conclusively demonstrated the presence of tuberculin in the exudations in many doubtful cases. Secondly, in many instances post-mortem examination of patients in whom acute pleurisy has arisen suddenly without previous apparent ill health, when death has taken place from some cause early in the course of the affection, has demonstrated the presence of pulmonary tubercle. Furthermore, although the exact result of tracing the after histories of cases of acute pleurisy by different observers naturally differs to a certain extent, there is a general agreement that a considerable proportion—from 30 to 40 per cent.—die of tuberculosis. Thus it will be seen that the evidence in favour of the tuberculous origin of many cases of so-called idiopathic acute primary pleurisy is very strong. It is not unlikely that a more systematic examination of the previous and after-histories of each case might even establish the percentage of tuberculous pleurisies as high as 70 to 75, the proportion arrived at by certain French observers. If this should prove to be the case, instances of idiopathic primary pleurisy must be few and far between, especially as in certain of the non-tuberculous cases other species of micro-organisms, to be presently mentioned, have been isolated from the inflammatory exudations. It should be remembered, too, that when cases are proved to be tuberculous in origin, they do not, strictly speaking, belong to the category of primary pleurisies, insomuch as the affection is in the vast majority of cases secondary to the deposit of tubercle in the lung or elsewhere.

Secondary pleurisy may arise in the course of many affections, both general and local. As the most familiar instance, we may mention the pleurisy which invariably accompanies acute lobar pneumonia. As the cause of this form of pneumonia is now generally thought to be the pneumococcus pneumoniæ (of Talamon and A. Fränkel), this micro-organism must also be looked upon as the exciter of the pleurisy which accompanies the lung inflammation. Some cases have been described in which pleurisy alone has occurred from pneumococcal infection, without the usual pneumonia. In such cases the pleurisy would have to be considered as a primary affection. Pleurisy may complicate scarlet fever, diphtheria, typhus fever, puerperal fever, smallpox, pyæmia, and the like, but whether it is to be in each case attributed directly to the specific microbe which may be presumed to have caused the original affection or not, cannot be definitely stated. Secondary pleurisy also may occur in rheumatic fever, and in acute and chronic Bright's disease, but the cause of its so arising is not at present clear; when, however, it complicates the end of various chronic diseases, such as cirrhosis of the liver, chronic Bright's disease and cancer, Osler suggests that it may be tuberculous in origin. As regards the secondary pleurisies, which are local to all appearance, traumatic pleurisy may arise from blows on the chest, even without any fracture of ribs, but is naturally more likely to follow the latter, which indeed, among the civil population, is the most usual form of injury to the chest; although bullet wounds, stabs, and the like, sufficiently common in military surgery, occasionally take place. In such cases, however, the pleurisy is likely to be complicated by the effusion of blood into the pleural cavity. The entrance of pus from the penetration of an abscess in the neighbourhood, pulmonary, hepatic, splenic, or abdominal, also sets up what may be considered a traumatic pleurisy. The rupture of a blood vessel, or the leaking of an aneurysm into the cavity, might also, if it did not at once produce fatal hæmorrhage, set up pleural inflammation. Perhaps the most frequent of all varieties of local pleurisy is that due to the presence of tubercle in the subjacent lung; and, indeed, it may be safely affirmed that hardly a case of pulmonary tuberculosis escapes a greater or lesser amount of pleural inflammation. Pleurisy is wont to accompany gangrene and new growths of the lung, as well as pericarditis, new growths in the mediastinum, aneurysms of the aorta, and also, in some instances, enlarged bronchial glands.

Pleurisy may attack either sex, but the male, as being more exposed to alterations of temperature, is somewhat more prone to the affection; and at any age, with the doubtful exception of very young children or infants. Occupation appears to have no specific action in its production. The left side is rather more frequently attacked than the right, the side more exposed to cold than the opposite, and the lower than the upper part of the pleura. Any condition lowering the general health, such as mental anxiety, insufficient food, alcoholism, insanitary conditions, and the like, appears to predispose to pleurisy.

Pathology and morbid anatomy.—As inflammation of the pleura occurs in different degrees, so the amount and composition of the exudation poured out, as well as the extent and distribution of the inflamed parts, vary greatly. Either the visceral or the parietal pleura may be attacked, but most commonly both of the pleural surfaces are implicated.

The first change which occurs in a serous membrane during the process of inflammation appears to be an engorgement of the blood vessels over the inflamed focus; with this engorgement there is more or less exudation of lymph, with or without punctate hæmorrhages into the membrane. At the same time the endothelium covering the serous surface becomes cloudy, and the cells proliferate. In the human subject the inflammatory process may begin in the pleura at one or more foci, and when there are several foci there is tendency for them to coalesce by spreading. The intensity of the exudation in all probability depends upon the nature and severity of the irritation, and upon the condition of the body attacked.

The amount and nature of the exudation have been used, as before mentioned, as a basis for an anatomical classification of pleurisies which is often employed for purposes of description. While acknowledging the convenience of this classification, it should be remembered that it is a purely arbitrary one, and that each class tends to run into the next. The four classes are as follows:—(1) Pleurisy with scanty effusion (dry or adhesive): (2) pleurisy with abundant fibrinous effusion; (3) pleurisy with sero-fibrinous effusion; (4) pleurisy with purulent effusion.

It will be seen that the first two classes could be included under the head of pleurisy with fibrinous effusion (scanty or abundant), and also that the classes 1, 2 and 3 might be called pleurisy with non-purulent effusion as contrasted with class 4, pleurisy with purulent effusion. Before proceeding to the description of the different classes, it will be as well to consider shortly the chief facts which have been collected, showing the relation of micro-organisms to the causation of the disease. This subject has of necessity been mentioned incidentally in the preceding paragraphs. It will be as well in this connection to deal first of all with purulent pleurisy.

The presence of micro-organisms in purulent exudations is practically constant, although the microbes are not the same in every case. Those which have been most frequently isolated are:—*Pneumococcus* or *diplococcus*, *Streptococcus pyogenes*, *B. tuberculosis*, *Staphylococcus pyogenes*; but other species have also been found, *e.g.* saprophytic microbes, *B. typhosus*, bacillus of influenza, Friedländer's pneumo-bacillus, and so on. Each species is not, however, always isolated pure from these exudations; sometimes two or more are associated. Thus the pneumococcus may be associated with streptococcus and staphylococcus pyogenes, as may also the *B. tuberculosis* with these or with saprophytic micro-organisms. The very constant appearance of micro-organisms in purulent pleurisies makes it highly probable that it is to their presence the condition is due. This is all the more likely as the organisms most frequently isolated are those known by experiment to be inciters of inflammation and pus producers. Thus the pneumococcus of Fränkel is esteemed "as the principal exciter of inflammation of infectious nature in the human body"; the streptococcus pyogenes and the staphylococcus pyogenes are some of the chief pus producers, and the tubercle bacillus has also been demonstrated as having a similar property.

Considering the non-purulent pleurisies together, it must be confessed that the positive evidence in favour of their bacterial origin is not so conclusive. It is known, however, as was mentioned in the preceding section, that the tubercle bacillus is present in a certain proportion of sero-fibrinous cases; in some others, streptococci, staphylococci or pneumococci have been demonstrated, but as a rule the exudations, according to experiments which have been made so far, have proved to be non-bacterial. We know little about the presence or absence of bacteria in cases of localised or dry pleurisy, or, indeed, whether the irritation of a growth in the lung, for example, is capable of causing pleural inflammation of itself without any other factor entering in; it is not even certain whether the tubercle bacillus is the direct cause of the pleurisy which follows the presence of superficial tubercle in the lungs, although this is highly probable.

To summarise, then, we may say that pleurisy with purulent exudation is almost certainly of bacterial origin, and that a number of cases of non-purulent pleurisy may also be included in the same category, while it is not at present possible to deny that some cases may be of non-bacterial origin. It is clear, however, that there is no one specific micro-organism which produces pleurisy; many species may set it up, but the *B. tuberculosis*, the *Pneumococcus* and the *Streptococcus pyogenes* more frequently than others.

As a kind of rider to the problem as to the bacterial origin of pleurisy, is the question, which strictly speaking belongs to the ætiology of the disease, as to the part played by catching cold or a chill on the one hand, and by trauma on the other, when either precedes the appearance of pleurisy. Are they exciting or only predisposing causes? Without laying down any absolute dogma on the subject, it may be said that the trend of opinion is in the direction of regarding them as only predisposing causes; a chill acting by producing a general effect upon the organism, and trauma by producing a local, and perhaps a general effect, sufficient to diminish its power of resistance to the attack of the micro-organisms, which are only waiting their opportunity.

Dry pleurisy, pleuritis sicca, adhesive pleurisy.—In this variety the inflammation is generally confined to small foci, and the exudation is very scanty, in some instances hardly appreciable. At other times the exudation, though slight in amount, is of definite fibrinous nature. Adhesion of the two surfaces may follow here and there; this often happens in lung tuberculosis.

Pleurisy with fibrinous effusion.—A typical example of this variety of pleurisy is seen in connection with lobar pneumonia (pleuro-pneumonia). In it there is exudation at various foci, which tend to run together, of highly coagulable lymph containing a variable number of white blood corpuscles. The latter, by breaking up, set free the fibrin ferment which is necessary to the coagulation of the lymph, and the fibrin is deposited upon the pleural surfaces in layers of different thickness. The serum resulting from the coagulation is scanty, and for the most part is contained in the meshes of the fibrin. On post-mortem examination of such cases, the visceral and parietal surfaces of the pleura may be completely covered with rough and even shaggy deposits of a yellowish white colour, and shreds or bands of the same material are seen passing from one surface to the other. These shreds or bands are often very soft and semifluid, from being soaked in or from containing the serous fluid which results from the coagulation of the lymph. It seems probable that the exudation does not occur all at once, but that the deposit may be built up layer upon layer.

The exudation takes place into the scanty subserous tissue as well as upon the surface of the pleura itself; consequently, in some cases, the thickness of the inflammatory layer may be very considerable, in extreme examples being upwards of half an inch.

The changes which take place in the fibrinous exudation may result in complete recovery, by the immediate absorption of the fluid portion, and the subsequent absorption of the solid material. Such a termination may be hoped for in healthy persons in whom the disease is not excessively severe, and in whom absorption begins at an early period. The absorption is said to take place chiefly through the lymphatics. In incomplete recovery, with a more or less complete adhesion of the two pleural surfaces, by the gradual organisation of the exudation into connective tissue. Part of the fibrinous material, however, may remain unorganised between the thickened surfaces of the pleura. The thickened pleura may also after a time undergo more or less complete calcification (sometimes called ossification). The obliteration of the pleural cavity, and the adherence of the lung to the diaphragm, must of necessity produce changes in the appearance of the chest, and to a certain extent in the position of the thoracic organs, to which attention will be directed later. A pleurisy beginning in the fibrinous form may, under unfavourable circumstances, become purulent. A fibroid condition of the lung is an occasional after-effect of fibrinous pleurisy. It is exceedingly rare for death to result directly from this form of pleurisy.

Pleurisy with sero-fibrinous effusion.—Sero-fibrinous pleurisy shades off into the preceding form, but in typical cases differs materially from it. The effusion from the first is less coagulable or contains a smaller proportion of the fibrin-forming materials and fewer corpuscles, so that the deposit of fibrin, although present upon the surfaces of the pleura, is never a prominent feature of the inflammation. It would seem almost as though the severity of the process is less than in the preceding form; the fluid effused being a more dilute lymph. The focus of inflammation may vary in position as well as in area, but the exuded fluid always collects in the lowest part of the pleural cavity, which varies with the position of the patient. As the patient generally lies upon his back, the fluid gravitates to the lowest part behind, not at first compressing the lung, which is pushed away from the chest wall by the separation of the visceral and parietal surfaces of the pleura, and as it were floats upon the surface of the fluid, as far as its more or less fixed condition will permit. Later on, as the effusion increases, the lung may become compressed against the spinal column, and in extreme cases is airless; at the same time the pleural cavity, being greatly distended, pushes to the opposite side the mediastinum and its contents, and depresses the diaphragm, and with it the abdominal contents, *e.g.* the liver, if the effusion is on the right side, and the stomach and spleen, if it is upon the left. The quantity of fluid contained in the pleura in such cases varies greatly. Two, three, or four pints have been withdrawn by a single aspiration, and this amount may be greatly exceeded.

The fluid itself is yellowish or greenish, and more or less clear, sometimes containing shreds of fibrin; it is alkaline, of specific gravity from 1016 to 1023, and highly albuminous, containing serum globulin and serum albumin as well as salts, of which sodium chloride is the chief. It appears, therefore, to be practically a more or less dilute serum. The deposit upon both surfaces of the pleura is yellowish white, and as a rule thin. Sometimes the fluid is found to coagulate after removal from the chest. Considerable differences are observed when serous effusion occurs in a pleural cavity, partially obliterated by previous inflammation and adhesion. In such cases the position of the fluid and the direction of its spread are governed by these adhesions, and the fluid may be confined irregularly in cavities formed by them,—loculated or encapsuled effusions result.

Serous pleurisy or pleurisy with effusion, although it may occur under other conditions, is most commonly of tuberculous origin. Cases of serous effusion may terminate in—complete recovery, when absorption of the fluid takes place quickly, the deposit upon the lung not having become organised, a due expansion after the decrease of the fluid is possible. The deposit also upon the pleural surfaces may be entirely absorbed, and the displaced organs resume their natural position; incomplete recovery, with a partial expansion of the compressed lung, is often the best that can be expected. Too often the absorption of the fluid is slow and prolonged, in which case the lung is exceedingly unlikely to resume its former dimensions, the organised deposit upon it being a great hindrance. As a consequence of the non-expansion of the lung as the fluid decreases, the thoracic cavity has a tendency to diminish, the chest walls fall in, the intercostal spaces are narrowed, the diaphragm is drawn up, and the abdominal organs encroach upon the thoracic cavity. The heart is drawn, by the contraction of the fibrous adhesions, towards the affected side, and with the rise of the diaphragm the apex beat is sometimes found in the third, or even the second interspace; the shoulder of the affected side drops, the side moves imperfectly in respiration, and the spinal column may become more or less curved towards the affected side. Sometimes the thickness of the pleura remains for years an inch or upwards. Fibroid phthisis, from the development of fibrous tissue in a lung which has been compressed by the fluid, is by no means rare. Bronchiectasis may also occur as a sequence of the compression of the lung. The fibrous tissue which produces these effects seems to arise from a low form of interstitial pneumonia. Serous effusions may sometimes become purulent. Pleurisy with serous effusion may occasionally terminate in death; in acute cases, from compression of the lung interfering with due aeration of the blood; from compression of the heart producing fatal syncope, or from engorgement of the right side of it from obstructed pulmonary circulation. From this latter cause acute fatal œdema of the lung may occur. Death may also result indirectly from empyema or phthisis.

Pleurisy with purulent effusion (*Empyema*).—In this variety of pleurisy, commonly termed empyema, the effusion which takes place, although possibly richer in colourless corpuscles from the first, is probably not always at once purulent. Micro-organisms are always present. It would seem that the exciting inflammation must be severe, producing a condition of the blood vessels by which the corpuscles pass through their walls with unusual facility. In the effused fluid these corpuscles speedily multiply, then die, and pus is produced. The effusion is generally thick and creamy, of a greenish yellow colour, very viscid, and, on standing, separates into two layers, the lower layer of corpuscles and the upper of liquor puris. It may, however, be thin, pale, and watery. It is generally odourless, and alkaline in reaction. The purulent effusion does not, as a rule, contain any shreds of fibrin, and it is thought that if fibrin is formed, it undergoes resolution. The deposit upon the pleural surfaces is comparatively thin, and consists chiefly of precipitated corpuscles. Purulent pleurisy may terminate in complete recovery. This, however, is not so likely to occur as in the serous variety, and very seldom happens, unless the empyema is evacuated early. In rare cases it is said that the fluid portion of the pus is absorbed, and that the corpuscles undergo caseation and subsequent absorption. This termination, however, must be considered very uncommon. Pneumococcal empyema is the most favourable variety.

It may also terminate in incomplete recovery. If the empyema remains undisturbed for some time, it tends to point, and may be discharged by perforation of the chest wall. The usual position for this pointing is in front of the chest at the fifth interspace, close to the edge of the pectoralis major (Marshall); it may also open higher than this, or, indeed, at any position as low down as the pleural cavity extends. It may, by tracking through the muscles, point in almost any part upon the surface. It may open through the lung tissue and be discharged through the bronchus and trachea, or it may find its way into the neighbouring serous cavities, the pericardium or peritoneum.

Finally, empyema may terminate in death, either from the causes enumerated under the head of serous pleurisy, asphyxia, or syncope; from long-continued hectic fever, or from pyæmia or lardaceous disease; also by its bursting into the pericardium or peritoneum, from pericarditis or peritonitis.

Hæmorrhagic pleurisy.—Some would add to the four classes of pleurisies already treated of, a fifth, namely, hæmorrhagic pleurisy, a variety in which the effusion is blood-stained to a greater or lesser degree. It seems, however, unnecessary to do this. Blood is found in the effusion under several conditions, namely—when primary pleurisy is attended with great hyperæmia, so that rupture of or oozing from the blood vessels occurs (which is very rare); when some newly-formed blood vessels in the organising fibrinous deposit give way; in such affections as cancer, Bright's disease, or cirrhosis of the liver; also in malignant fevers, in which there is great asthenia. The explanation of the hæmorrhagic exudation in these cases is very difficult. In rare cases of hæmophilia and scurvy.

To conclude the section on the pathology of pleurisy, one other point of interest may be mentioned; in some instances, fluid accumulates, over and over again, in the pleural cavities after removal. As regards the purulent effusion, it is not, of course, uncommon for a so-called pyogenic membrane to be formed, which would account for the phenomenon; but with respect to the serous effusion such a reaccumulation is more difficult to explain. In the latter case, however, the fluid becomes more and more diluted, and approaches in composition to the fluid of hydrothorax, and the condition is, indeed, hardly to be distinguished from a dropsy of the serous membrane.

Symptoms.—In describing the clinical phenomena of pleurisy, it will be as well to consider those of the four chief anatomical forms seriatim. In **pleuritis sicca** there is naturally but little constitutional disturbance, and the chief, and probably the only, symptom is a pain in the chest over the situation of the inflamed patch. Such pains are very frequent in tuberculous disease of the lung, and though sometimes severe, are seldom of long duration.

Fibrinous pleurisy may be merely an exaggerated form of the foregoing, the only difference being more extensive exudation; as such it occurs in lung tubercle, cancer, abscess or gangrene, giving rise to pain in the side or elsewhere, and more or less fever with perhaps dry cough. These symptoms may be of short duration. With the symptoms there is impaired resonance, harsh breathing, and a friction rub of more or less extensive area on physical examination. More frequently fibrinous pleurisy is secondary to pneumonia (acute lobar), and may be considered part of the pneumococcal infection. Under such circumstances it may appear at the beginning, which is most common, or at a later period in the course of the affection. Its symptoms, with the exception of the pain in the side, cannot be distinguished from those of the pneumonia of which it forms a part. Occasionally fibrinous pleurisy is primary, and, if then due to pneumococcal infection, gives rise to symptoms which are almost exactly like those of pneumococcal pneumonia, may be accompanied by labial herpes, and defervesces by crisis. It should be noted, however, that primary pneumococcal pleurisy is usually accompanied by sero-fibrinous, and very often by purulent effusion.

Sero-fibrinous pleurisy is the most usual form of primary pleurisy, which arises suddenly in the apparently healthy as an acute affection with or without previous history of a chill. As we have seen, it is very frequently tuberculous in origin, but is also associated with other micro-organisms (pneumococcus, streptococcus, and the like). This affection presents in the most typical form the cardinal symptoms of pleurisy, viz. pain in the side with fever, dry cough and interference with respiration. It will be best to consider some of the chief symptoms separately.

It seldom happens that the disease is ushered in by any well-marked rigor, but shivering of greater or less severity may occur, and may also recur for several days after the commencement of the disease. In children, headache, vomiting, or convulsions may represent the rigor. The temperature, except in specially severe cases, is seldom higher than 102° F. There may be more or less constitutional disturbance, with headache, rapid pulse, 100 or 120 per minute, furred tongue, pains in the back and loins, and febrile urine, which is concentrated and high-coloured. It should be remembered, however, that these symptoms may be almost entirely absent, and the temperature may be only a degree or so above normal. Again, the symptoms of fever may very quickly subside, or may remain more or less marked for some time, even weeks; or the temperature, rising for the first three or four days, may continue at the maximum, with slight variations, for a week or more, with slight morning remissions, and may then tend to gradually fall to normal in the morning, with an evening rise of 2° or 3°. If the raised temperature continues for more than two or three weeks, and its maximum exceeds 102°, it is almost certain, either that we have to do with tuberculous pleurisy, or that the effused fluid is purulent.

A sudden pain or stitch in the affected side is generally present in pleurisy. It is increased by taking a deep breath, and may prevent any but the shallowest respirations; it is also increased by movement, and by pressure, unless firm, evenly applied, and well-sustained. Generally speaking, the pain is very acute and stabbing. Although confined to a definite area, it is seldom localised to a small spot, and may be very diffused, being sometimes marked, even at a distance from the seat of the lesion. It is occasionally felt on the opposite side of the chest, as well as upon the side of the pleural inflammation; it may be felt in the shoulder, axilla, over the sternum, or beneath the clavicle, and when the diaphragmatic pleura is affected, over the cartilages of the false ribs. As the effusion increases, the pain diminishes or disappears. It may return as the effusion subsides, but is not then of such a severe stabbing character as before. It is said that the pain lasts longer, and may be more severe, in cases of tuberculous pleurisy, and in cases of purulent effusion.

It should be recollected, however, that the pain is not always well marked—as, when the effusion is slow, or the condition producing it chronic, it may be only very slight, and insufficient to call attention to the new condition. It is also slight, and sometimes absent, in cases of very young children, in old people, and in the insane. It has, however, been calculated that pain, of greater or lesser severity, occurs in about 85 to 90 per cent. of all cases.

From the first the respirations are almost certainly increased in number, and are shallow in character, averaging from 25 to 30 per minute. The shallowness of the respirations may be due to the pain which a deeper breath causes, and which the patient tries to avoid by breathing incompletely. Later on it is due to the fact that one lung has to do the work of two. The febrile movement adds to the rapidity of the respirations. In severe cases, and when the effusion is very excessive, the dyspnœa may be still greater, with laborious respiratory efforts, in which the extraordinary muscles of respiration are called into play, and there are indications of insufficient aëration of the blood, shown by blueness of the lips, and coldness and blueness of the extremities. If the condition remains unrelieved, the patient gradually becomes asphyxiated. In ordinary cases, however, there is remarkably little dyspnœa as long as the patient is undisturbed and quiet, even if the fluid in the chest is considerable.

Cough, generally of a dry, harassing character, is commonly present. It is ineffective, and adds greatly to the pain. If there is, at the same time some amount of bronchitis, or congestion of the lung, the cough is accompanied by more or less frothy expectoration.

The patient is seldom able to lie in any position indifferently. As a general rule, at first it is impossible for him to lie upon the affected side, as by so doing the pain is increased, and he must lie upon his back, or towards the sound side. When the effusion separates the two surfaces of the pleura, this is no longer the case; the patient can lie upon either side, but, later on, if the accumulating fluid is considerable, he lies towards the affected side, in order to take off the pressure from the sound side. Occasionally we meet with cases of pleurisy with considerable effusion, in which there is no history of inconvenience of any kind in any marked degree.

Purulent pleurisy may occur as a primary pleurisy, apparently from the same causes as sero-fibrinous, and the difference in the exudation is generally to be attributed to the condition of the patient. Thus it is wont to replace the usual form of exudation when the patient is in poor health, and is the form of acute pleurisies often found in children. It may also arise, we may presume, from a more virulent infection. It is frequently associated with lobar pneumonia, especially at the end of the affection; with scarlet fever, typhoid fever, and occasionally with other infectious fevers as a complication. It is sometimes tuberculous, but not so often as the preceding variety. It is the form especially likely to arise from penetration of the pleura by malignant disease of the lung or œsophagus, or by tuberculous vomicæ. Traumatic pleurisy may also be purulent. Sero-fibrinous pleurisy may be converted into empyema by the introduction of atmospheric germs in the operation of tapping the chest.

When the fluid within the chest is purulent, there may be, in addition to or modifying the preceding symptoms, the following:—Rigors or repeated shiverings occurring some days after the first appearance of the pleurisy; distinct hectic fever, the temperature chart being marked by regular rises of several degrees in the evening, and corresponding falls in the morning to about normal. Night or sleep sweats, harsh skin, increasing emaciation, and if the case is allowed to go on without operative interference, clubbing of the fingers. The appearance of a localised redness and œdema of the surface, with the skin shining and glazed over the swelling, and a sense of fluctuation on the application of the fingers, indicate the pointing of the purulent collection, and render the diagnosis of empyema certain. It need hardly be said, however, that if possible the diagnosis of empyema should be arrived at before the pointing appears. The position of pointing is generally on the front of the chest, even as high as the second or third interspace, most commonly, as before mentioned, in the fifth.

Physical signs.—When the effusion is confined to a small area of the pleura and the exudation is scanty, in other words, in pleuritis sicca, the most common sign is a grating sound, "friction rub," heard at the end of inspiration. This sound is not, however, invariably present. It may or may not be accompanied by a friction fremitus on the application of the hand and slight impairment of resonance on percussion. The sign or signs are localised to one particular spot.

When the effusion is not localised to any one spot in the pleura, but is general throughout the cavity and fibrinous in character, very small amounts of serous fluid being present, the signs are very much more marked. The affected side moves much less freely than the sound side. The percussion note is impaired. On auscultation, the breath sounds are feeble, or there may be tubular or bronchial breathing. Together with these signs, there is almost always a friction rub to be heard in one or more situations, most commonly at the lower part of the pleural cavity, where the costal and diaphragmatic pleural surfaces are in contact. Sometimes instead of the friction rub there is a creaking or crepitant râle, which is very similar to the râle heard in tuberculous disease of the lung or in catarrh of the finest bronchi.

When the effusion is more serous than in the second variety, and when it increases in amount so as to become considerable, we have to do with the physical signs of pleurisy with effusion. At first, when the effusion is beginning, there may be, but only in some instances, the signs of the previously described variety, but as it increases the friction sound or râle, and the friction fremitus disappear.

On inspection, when the effusion is well established, the movement of the affected side in respiration is diminished, especially of the lower part of the chest, the difference between the two sides being especially evident when the patient takes a deep breath. The affected side appears to be larger and fuller than the sound one, and this difference may be measured by the tape or by means of a cyrtometer. Sometimes there is a difference of an inch or more between the two sides. The intercostal spaces are obliterated, but if there is ever bulging, this must be a rare occurrence. The apex-beat of the heart may be seen to be displaced towards the sound side; if the effusion is on the right side, it is dislocated outwards, sometimes well beyond the left nipple, sometimes even into the axilla, or upwards into the third or fourth interspace. When the effusion is on the left, the apex-beat may not be visible, being behind the sternum, but when the effusion is very great it may be seen beyond the sternum as far as the right nipple, and sometimes high up in the third or fourth intercostal space.

On palpation, the displacement of the apex-beat may be more accurately examined and localised. Fluctuation is hardly ever to be demonstrated, and the same remark applies to pulsation. The vocal fremitus is generally greatly impaired or entirely absent over the area of the pleural exudation. This sign, although not absolutely constant, is one of the most important which can be obtained, and by careful palpation it is possible to follow the increase or diminution of the pleuritic fluid. In some cases, particularly in children, fluid may exist in considerable amount without the fremitus being markedly diminished.

On percussion, there is dulness with increased resistance over the area of the fluid. The dulness almost invariably begins at the posterior and lowest part of the chest, and as the effusion increases, generally speaking, the extent of dulness is always greater behind than in front—that is to say, if the patient is lying down in bed, as is usually the case—and a considerable amount of dulness may exist behind, and fairly good resonance in the axilla and in front. In small effusions with the dulness at the lowest part of the chest behind, there is good resonance above, and even when the dulness behind rises above the level of the angle of the scapula, there may be no appreciable dulness in front, when the patient is in the supine position. The degree of dulness over even large effusions is not constant, being at one time greater than at others, and the resonance of the chest is frequently impaired for some little distance above the level of the fluid. The effusion may be so great as to distend the whole of the pleural cavity, and in that case the dulness is found in front to the level of the first rib, and as the mediastinum and its contents are pushed over to the opposite side, as a general rule under these circumstances, the dulness extends to the opposite edge of the sternum, and sometimes even beyond this limit.

It should be noted that the statement so often made in text-books to the effect that the fluid in cases of pleurisy, and consequently the dulness on percussion, vary with position, can very seldom be proved correct, and the slight alteration which occasionally takes place from this cause requires skilled percussion for its demonstration. The upper limit of the fluid when the patient is erect never seems to be a straight line, but is a curve, the highest point of the curve being in the axilla; and it is a fact to which great importance is attached, that even in the case of excessive effusions there remains an area in front below the clavicle, more or less triangular in outline, over which the percussion is tympanitic in resonance (skodaic resonance), and a corresponding area of similar resonance exists behind. The dulness behind close to the spine is never absolute.

On auscultation, the breath sounds over the area of the exudation are diminished or absent. Sometimes there is tubular or distant bronchial breathing, occasionally there is in addition a somewhat metallic quality in the sounds, which are even amphoric or cavernous in rare instances. These abnormalities of the breathing are more often present in cases of children, and are apt to be confusing. At the upper limit of the fluid the breathing sounds are usually harsh, and may be tubular or bronchial. This is especially the case over the area where the percussion is skodaic. As a rule the vocal resonance is diminished or absent over the area of fluid, but sometimes the voice may be exaggerated and bronchophonic in character. The well-known twanging or nasal character of the voice, likened to the voice of a goat, ægophony, or the voice of Punchinello, is sometimes to be noticed at the upper limit of the fluid behind, but this sign is not by any means invariably present.

When the effusion has reached its height it gradually subsides, and the physical signs diminish. The most important indication of the gradual subsidence is to be obtained by careful examination of the diminishing area of dulness, and by noting the gradual return of the heart's apex-beat towards its normal position. On careful percussion, the lines indicating the upper levels of dulness, as the effusion diminishes from day to day, are found to be curved and not straight, as one would perhaps have expected. Somewhat elaborate statements have been made as to the order in which the fluid is absorbed from different parts of the pleura, but this order is not, according to our experience, by any means constant. With the improvement in the resonance the side diminishes in circumference, and the intercostal spaces resume their normal appearance. As the pleural surfaces tend to come together again, the friction rub or râle may be heard. The last place for the fluid to leave is the lateral region.

When the effusion is purulent, the physical signs are those of pleurisy with effusion. Until the empyema tends to point, however, there is no sure diagnostic criterion which distinguishes the nature of the fluid. No one, however, who has any suspicion that the fluid in the chest is purulent would think of waiting for it to point, but would at once proceed to an exploratory puncture of the pleural cavity, by means of a syringe fitted with a stout and sufficiently long needle, and to withdraw some of the fluid for examination. A morphine syringe will, on occasion, be suitable for the purpose. By this procedure it will be possible to arrive at a correct diagnosis.

Localised pleurisies.—As may be inferred from the account of the pathology of pleural inflammation given in a preceding section, pleurisy may be localised to different areas, may present physical signs confined to these areas, and may also show some modification in symptoms from those of general pleurisy. This is especially the case when fresh inflammation attacks a previously inflamed pleura, and the effusion is confined within certain limits by adhesions. As a general rule, when these loculated and localised pleurisies become important, from giving rise to special symptoms or physical signs, the effusion is found to be purulent, and for the sake of description we shall include all such cases under the head of localised empyemata, simply remarking that in some instances the effused fluid may be serous or fibrinous.

Collections of pus may be found loculated or encapsuled in different parts of the pleural cavity, but particularly between the lung and the diaphragm (diaphragmatic empyema), in the axillary region, in the front of the chest, or between the lobes of the lung. Sometimes from the position of the adhesions there may exist at one time several of these encapsuled collections of pus, or more rarely one or more of them may exist upon either side of the chest. These localised empyemata may tend to point.

When there is a collection of pus between the lung and the diaphragm, it is usually more marked behind than in front, is generally unilateral, and is most frequent in women of adult age. It may be associated with hepatitis or with subdiaphragmatic abscess or with wounds of the chest or abdomen.

It is not usual for this form of pleurisy to be separately diagnosed, but there are certain indications which should allow of this being done.

The pain is often of intense character, especially on cough or on any movement, extending over the whole of the hypochondrium, radiating in all directions, with great tenderness on pressure, particularly over the epigastrium, in the lowest interspace posteriorly, along the course of the phrenic nerve, and at a spot which has been called the diaphragmatic button, a spot one or two fingers-breadth from the middle line of the body in front on a level with the tenth rib. In addition to pain there is more than usual dyspnœa, and indeed orthopnœa with inclination of the body forward. The respiration is purely costal, and is shallow and rapid. The countenance is anxious and the cough frequently dry and painful. In addition to these symptoms the following may occur—delirium, hiccough, pain on swallowing, risus sardonicus, nausea, vomiting, and jaundice. These empyemata may ulcerate through the lung and be discharged through the trachea, or they may perforate the diaphragm and produce fatal peritonitis.

The condition of the chest in other cases of localised collections of pus varies. There may be a local bulging, or the bulging of the chest wall may be above the situation of the pus. The former is usually the case when the empyema is in the axilla. The movement of the affected side is probably diminished, but the heart is not, as a rule, displaced; usually there is dulness on percussion, not altogether localised to the position of the pus, as the neighbouring part of the lung may be considerably collapsed. Respiration is feeble, or there may be tubular or bronchial breathing.

Under certain conditions, the pulsations of the heart are communicated to the chest wall by the fluid of pleurisy, but particularly when the fluid is purulent. The pulsation is found all over the affected side, or it may be felt over the cardiac area, or where the empyema tends to point. The extreme distension of the pleural cavity with fluid appears to be the cause of this phenomenon, since, on the withdrawal of some of the fluid, the pulsation ceases to be observed. Pulsating empyemata, even when the fluid is localised, may be mistaken for an intrathoracic aneurysm; but the dulness on percussion, in a position different from the usual position of an aortic aneurysm, the absence of cardiac murmurs and the very faint sounds of the heart heard on auscultation, would help the diagnosis. Aortic aneurysm may, however, exist together with fluid in the pleural cavity.

Course.—The course of pleurisy varies greatly. If the pleurisy is of the dry or adhesive variety, the pain and difficulty in breathing are very temporary, lasting perhaps a few hours at most, but it should be noted that if the diaphragmatic pleura is affected the pain is more prolonged in duration.

As regards the fibrinous variety, supposing the inflammation is general, the acute symptoms do not last many days, and the same is the case with the serofibrinous variety of the affection. The fever, never very high, subsides in a week, or at any rate long before the effusion has been absorbed. Complete absorption of an extensive effusion cannot be expected under three weeks, and a much longer time, even extending to months, may elapse before the patient is really convalescent.

Sometimes a case which begins acutely with high fever may quickly subside,—never, however, in our experience by crisis, although this has been noted by competent authority,—with rapid reabsorption of the fluid. At other times an apparently favourable commencement may be followed by a very slow and interrupted absorption. These are simply examples of chronic stages of acute pleurisy. It seems, however, that there are chronic as well as acute varieties of the four forms of pleurisy we have been describing, not only in course but also in onset. Thus as regards fibrinous pleurisies, cases are sometimes seen in which, after death, extensive adhesions may be present with no previous history of pleurisy, and instances of sero-fibrinous and purulent pleurisies are occasionally met with of very insidious onset and chronic course.

When the fluid is for a long time unabsorbed, the patient commonly presents marked constitutional symptoms, he wastes and becomes anæmic.

Diagnosis. — Pleurisy may possibly be mistaken for many other affections both within and without the chest, or *vice versâ.* Of the former may be mentioned pleurodynia (intercostal neuralgia or myalgia), periostitis of the ribs, or abscesses of the chest wall, all of which, however, should present little difficulty since they are unaccompanied by alteration in the sounds heard on auscultation, and by only slight, if any, dulness on percussion of parts of the chest not corresponding with the usual seat of pleuritic dulness; whereas careful examination of the affected part will most likely indicate the nature of the affection.

Pleural effusion may be confused with enlargements of the liver, particularly with abscess or hydatid disease, and with enlargements of the spleen, as the enlarged viscera may, under certain conditions, encroach upon the thoracic cavity. The diagnosis is arrived at from the history of the several diseases, together with the physical signs in the chest pointing to pleurisy or the presence of hepatic or splenic tumours, to be made out by examination of the abdomen. It should be noted that splenic and hepatic tumours usually follow the movements of the diaphragm in respiration; whereas in the case of the pleural effusion, the diaphragm being fixed, no alteration of the abdominal organs occurs upon the affected side during respiration.

The chief thoracic diseases with which pleurisy is liable to be confused are lobar pneumonia, atelectasis, pulmonary tuberculosis, and new growths of the lung or mediastinum. To each of these it is necessary to give attention.

Lobar pneumonia comes on with marked rigor, high fever, and herpes about the lips, and its fever usually terminates with a distinct crisis. The cough is as a rule accompanied by rusty sputum. All of these signs, except the rusty sputum, may, it is true, occur in the rare cases of primary pneumococcal pleurisy. On physical examination of the chest, the intercostal spaces are neither flattened nor prominent, the apex-beat of the heart is not displaced, there is no friction fremitus, and the vocal fremitus over the affected part is increased; the dulness on percussion does not alter with position, and the upper limit is a curved line, following, as a rule, the fissures of the lung, is not so resisting or absolute, and never extends beyond the middle of the sternum. Whereas, with auscultation, there is the characteristic bronchial or tubular breathing, bronchophony and crackling in the early stage, and crepitatio redux when the inflammatory products are being absorbed.

Atelectasis of the lung is chiefly to be distinguished from pleurisy by the absence of fever and constitutional symptoms, by the position of the dulness if present, and its not being absolute or resisting, the absence of breath sounds, with bronchial or tubular breathing and bronchophony sometimes, and generally the presence of vocal fremitus over the affected area.

Pulmonary tuberculosis is most likely to be confused with pleurisy in the somewhat rare instances in which the lower lobe of the lung is affected without the upper part being tuberculous. When there is effusion, the absence of vocal fremitus, the displacement of organs, and the absolute resisting dulness, should be sufficient for the diagnosis; and, on the other hand, the presence of tubercle bacilli in the sputum, the increased vocal fremitus, the bronchial or amphoric breathing, with or without crepitant râles, ought to be enough to suggest the diagnosis of tubercle. In cases of fibrinous pleurisy, the alteration of the affected side and the contraction of the chest are sufficiently marked in chronic cases; and if the lung is in a fibroid condition, the absence of tubercle bacilli will point in the right direction. In cases in which the pleura is greatly thickened, without contraction of the side being present, the diagnosis is often of great difficulty, as vocal fremitus may be present, and sounds such as are heard over a vomica may be present as well; but the absence of fever, and of tubercle bacilli in the sputum, will help in the diagnosis. If a clear history of an acute attack of pleurisy can be obtained, so much the better. The close connection between all kinds of pleurisy and lung tuberculosis must, however, always be borne in mind.

New growths in the mediastinum or lung, or aneurysms of the aorta or innominate artery, may be mistaken for pleurisy. As a rule, in the case of new growths, a history of gradual dyspnœa, without fever, may be obtained; and the gradual extension of dulness, almost always from above downwards, may be noted. The area of dulness, too, is often irregular in shape. With new growths, however, the vocal fremitus may be diminished or increased, and the breath sounds are almost always absent, except at first. In aneurysms, the position of the dulness, the presence of a pulsating tumour, and signs connected with the vascular system, as well as pressure symptoms, are usually sufficient for the diagnosis.

It should, however, be remembered that aneurysms of the aorta and new growths of the lung or mediastinum may often, even when small and almost unrecognisable, give rise to pleurisy, with considerable effusion, in which case it is extremely difficult, if not impossible, to diagnose the cause of the effusion. If the fluid is removed by aspiration, however, the symptoms and signs of the primary disease become more apparent.

Exploratory puncture.—In concluding this section upon diagnosis, it should be again insisted upon that in any doubtful case the presence of fluid in the chest can be proved by the introduction of a fine needle, in connection with a syringe, into the pleural cavity, and the fluid, if present, may be withdrawn. Care, however, must be taken, first, that the needle is long enough to pass through a considerable thickness of inflamed pleura; and, secondly, that no air be introduced when the needle is removed.

Prognosis.—Primary pleurisy may leave behind it a perfectly undamaged serous cavity; there is generally more or less adhesion. This is not, however, *per se* either dangerous to life or health. Independent of the prognosis of the affection which set up the pleurisy, in secondary cases the points in favour of a good prognosis would be general good health, fever of a subacute form, soon diminishing, and a rapid absorption of the effusion. Points against a favourable prognosis would be excessive effusion, especially of purulent fluid; great displacement of organs, and pressure upon the heart or great vessels; persistent hectic, with wasting and general asthenia; cyanosis or dropsy, or other dangerous disease from any cause; the existence of complications, such as the bursting of an empyema through the lung, or into neighbouring organs or cavities. The rapid production of pus in a pleurisy is a bad indication; sometimes within a week or eight days the effusion is so great as to cause imminent danger of death, unless the chest is tapped. This form of empyema is sometimes accompanied by very high fever, temperature 105° F., or upwards. It is a rare form, and has been called *pleuritis acutissima.*

Treatment —The treatment of dry pleurisy has to be directed to the relief of the pain, which is sometimes altogether out of proportion to the gravity of the affection. This may be done either by preventing the movement of the chest, by strapping or bandaging;—strapping preventing the movement of the affected side, and firm bandaging preventing costal respiration altogether;—or by counter-irritation, by means of a linseed meal or a mustard poultice, a mustard leaf, painting with iodine or, best of all, by the application of a rapidly-rising blister. The blister, which need not exceed an inch in diameter, will usually relieve the pain in a short time. Instead of counter-irritation, leeches to the painful spot or cold compresses will sometimes be useful.

When effusion of some amount has taken place, the patient should be kept in bed, and if necessary, should be propped up by means of extra pillows, as the raised position is often the most comfortable. Our object in the treatment should be to relieve pain if present, to alleviate the fever and cough, and to get rid of the fluid in some way, either by procuring its absorption or by actual removal.

These indications may be met as follows:—The pain may be relieved in one or other of the ways above mentioned; the fever, although seldom prolonged in uncomplicated cases, may be treated by the employment of quinine or digitalis, or both combined. Venesection is occasionally useful, especially when there is evidence of pulmonary congestion. The cough may be relieved by the use of mild opiates, but it is seldom troublesome unless there is at the same time bronchial catarrh.

As regards the effusion, the more rapidly the absorption is brought about the better, and for this purpose it is usual to exhibit diuretics and purgatives. Of the former may be mentioned acetate of potassium, spirits of juniper, and decoction of broom tops; and of the latter, such drugs as jalap, scammony, and acid tartrate of potassium, and others producing watery evacuations. The administration of iodide of potassium internally, and the application of iodine liniment to the affected side, appear to be of service; as are also diaphoretics, such as ipecacuanha and the spirits of nitre. Some have advised the restriction of the fluids of the food.

If the effused fluid remains in the chest in spite of treatment, and does not appreciably diminish within a week or ten days, the question of removing it by operation has to be considered. The fluid should in all cases be removed if it is purulent. In exploratory puncture of the chest, care must be taken that the needle of the syringe is not too fine, as thick pus may not be withdrawn through a fine needle; also that it be long enough and of strong make, since sometimes the thickness of the walls is great, and the tissue through which it has to pass is considerably indurated. In acute pleurisies there is sometimes evident danger of death from syncope or asphyxia, in consequence of the pressure of the fluid upon the heart or lungs. If the affected side of the chest becomes much greater than the sound side, and the subclavicular skodaic resonance almost disappears, while at the same time there is evidence of displacement of the diaphragm downwards, we may assume this condition to have arisen,—a condition of what is called positive intrathoracic pressure. When the absorption of the fluid is slow, and the patient's health is suffering in consequence, there is no arbitrary limit of time which should be allowed to elapse before operation is resorted to; it probably depends upon the circumstances of each case; but if the patient seems in any way failing, the operation should be done without delay. A fortnight will generally be sufficient to demonstrate this. In all cases of symptomatic pleurisy, when the effusion appears to add to the distress of the patient, already very ill, the chest should be tapped. A longer time may be allowed before tapping the chests of children and young people, as sometimes the fluid is absorbed, even when in excessive amount, if they are placed under favourable circumstances.

Tapping the chest.—*When the fluid is not purulent.*—Paracentesis is usually done by means of a cylindrical needle or fine trocar and cannula in connection, by means of an indiarubber tube, with an exhausted aspirator bottle. When the passage from the pleural cavity into the aspirator bottle is free, and the connection of the bottle with the outside air is stopped by turning the stopcock, the suction power of the partial vacuum assists the flow of the fluid. To obviate the use of the aspirator bottle, the tube in connection with the cannula may be carried into a suitable vessel half-filled with water, placed below the level of the patient's bed. By this method, when the fluid commences to flow out from the pleura under positive pressure, a syphon action is set up, which continues as long as the operator desires, and at the same time no air is permitted to pass up the tube. Some recommend incision instead of tapping with a needle, but in most cases this is not necessary or desirable.

When the fluid is purulent.—In all cases of empyema there is no alternative but free incision into the pleural cavity, although at the time of operation an exploratory puncture may first of all be done. The practice of resecting an inch of one or more ribs at the time of operation is very common at the present time. By these means a free exit for thick material from the chest is permitted, possible necrosis of the ribs from pressure of the drainage tube is avoided, and falling in of the chest walls is facilitated.

One of three situations is most commonly chosen for the puncture, namely—In the eighth interspace postero-laterally at about the junction of the anterior two-thirds with the posterior third. In this situation the muscular covering of the chest is thinner than elsewhere. In the sixth or seventh interspace (according to whether the operation is done on the right or the left side respectively), in or about the midaxillary line; this is the usual situation. In the fifth interspace, slightly anterior to the digitations of the serratus magnus.

When the chest is to be aspirated, it is unnecessary to anæsthetise the patient. He should be placed in a semi-recumbent position in bed, with the affected side near to the edge, lying with his head slightly raised. He should be directed to raise the arm on the affected side above his head; the point at which the puncture is to be made should then be marked. It should be in the middle of the interspace, to avoid the intercostal blood vessels. In the case of sensitive patients it may be as well to apply cocaine (20 per cent.) to the spot previous to puncture, and sometimes it is advisable to make a small cut before inserting the trocar. In the case of serous pleurisy it is not necessary or expedient to remove the whole of the fluid; some may remain behind, and will almost certainly be absorbed after the operation. When sufficient fluid has been removed, the surrounding skin should be pinched up while the cannula is being removed, and the puncture should be covered with thickly-spread strapping, or with a piece of lint soaked in collodion, and the chest bandaged. After the operation the patient should be kept quiet in bed and fed well. Careful after-examinations should be made to note to what extent the heart has returned to its normal position, and also to find out whether the fluid is reaccumulating.

In favourable cases of non-purulent effusion, and particularly those in which the operation has not been too long delayed, the results of tapping the chest are highly satisfactory. The anxiety of the patient is relieved, the heart and other organs resume their usual position either at once or very speedily, and the lung expands. The fluid remaining in the chest is absorbed, and no more is effused. Consequently, in a few weeks, there remain practically no abnormal physical signs to indicate the previous condition. In other cases, recovery almost as complete but more delayed may be expected, with or without some shrinking of the affected side. Sometimes, however, the fluid reaccumulates, and a second or third evacuation has to be undertaken. Ultimately, however, even such cases may recover, but there will probably be much shrinking of the chest, and other results of chronic pleurisy already enumerated. Sometimes the fluid on its reappearance is found to be purulent. In such cases the treatment which is adopted in treating empyema will have to be employed, namely, a free opening and drainage of the pleural cavity. The usual results of discharging empyema follow.

When incision of the chest has to be done to evacuate an empyema, and particularly when at the same time one or more ribs are to be resected, an anæsthetic is necessary. Under these circumstances, too, it is best to employ the lowest of the three possible positions of operation. The patient should not be turned over towards the sound side more than is absolutely necessary. The incision having been made either in the eighth space, or over the ninth rib, and the rib (or ribs) having been cleared for the space necessary, and about an inch having been removed with a pair of strong bone forceps, the pleura should be cut into with a scalpel, and for the sake of convenience the pus should be allowed to flow out through a cannula and tube into a suitable vessel. When as great an amount of pus has been discharged as possible, a stout drainage tube should be inserted (" the perfect tube " is a single, short, large-flanged rubber tube, without side-holes, with a bore of $\frac{3}{8}$ in. to $\frac{5}{8}$ in., long enough to reach the cavity without projecting far into it—S. Paget). Having secured the tube, so as to prevent the possibility of its being drawn into the chest cavity, the wound should be covered with a thick layer of antiseptic gauze, then of cotton-wool, and afterwards the chest should be firmly bandaged. The dressing not only receives the discharge, but prevents any great influx of air into the cavity. The daily washing out of the pleural cavity with some very weak antiseptic solution after the operation is strongly recommended by some, and as strongly condemned by others. In our practice we have altogether abandoned the use of carbolic acid and perchloride of mercury; and when washing out is done, either freshly boiled water is used, or a weak solution of boric acid.

When the operation is performed with due precaution, aspirating the chest is seldom complicated with any serious after-consequence, but syncope may occur. This may generally be obviated or arrested by the administration of brandy or some other stimulant. If fainting occurs, the operation should be abandoned for the time. Dyspnœa may also arise, generally speaking, shortly after the operation, with or without troublesome cough. As a rule, a change in the position of the patient will remove this, or the exhibition of an opiate or a morphine injection. Firm strapping of the affected side will often have a good effect. Hæmorrhage occasionally may be met with either from the puncture of a vessel, or from rupture of new vessels in the pleural membrane when the support afforded them by the fluid has been removed. Very rarely the lung has been punctured. Sudden death from shock, or less sudden death from acute œdema of the lung, has happened; and of the other untoward events which have been recorded, we may mention surgical emphysema, hæmaturia, and severe pyrexia. After the operation for evacuating an empyema, certain of the above complications may ensue, and the danger of shock is certainly greater.

Under favourable conditions, the discharge from an empyema diminishes and ceases in a few weeks, the wound in the chest wall healing up, and the thoracic contents resuming their normal position and functions, very slight deformity remaining; sometimes the discharge is very chronic, lasting for months, or even years, and possibly requiring further operative interference; but even such cases may take on a more healthy aspect, final recovery with greater or less deformity of the chest remaining.

In unfavourable cases the patient dies, either from the wasting discharge and asthenia, with or without amyloid disease of the internal organs (liver, kidneys, or intestine), or he may die from secondary pyæmic abscesses. In some cases these abscesses have been found in the brain.

Only temporary relief can be expected in cases in which the operation of paracentesis has been undertaken in order to relieve empyemata, secondary to a disease from which the patient is already "under sentence of death."

HYDROTHORAX.

Hydrothorax is a condition in which a greater or lesser amount of serous fluid is found in one or both of the pleural cavities. It is never a primary affection, and must not be confounded with the inflammation of the serous membrane. The fluid passes through the walls of the veins by transudation. The affections with which hydrothorax is most commonly associated are as follows:—Chronic diseases of the heart, both valvular and muscular, in which there is increasing dilatation of the right side and atony of its muscle arising from any cause. The fluid passes through the walls of the veins in consequence of the abnormal pressure which exists within them from general or local obstruction to the blood flow. Acute or more generally chronic diseases of the kidneys, in which the general composition of the blood is altered, being especially poorer in albuminous constituents. Neglecting the exceedingly rare cases in which a pure dropsy of the pleura is said to have arisen primarily from exposure to cold, or in the course of scarlet fever without albuminuria, and the clinically interesting but uncommon cases of chylous dropsy (chylothorax) from pressure upon the thoracic duct in any part of its course, it may be said that hydrothorax sometimes is seen in long-continued obstruction to the portal circulation combined with dropsy of the peritoneum. Of this condition cirrhosis of the liver is the most common cause. And, lastly, hydrothorax may arise in other conditions of the body in which the blood becomes hydræmic, *e.g.*

cachectic states from cancer, dysentery, leukæmia, and malaria, if sufficiently prolonged and severe.

The transuded fluid in composition differs considerably from that effused in pleurisy, in that it contains only about a third or less than the solids found in the latter (1·48 per cent. against 5·13), and less than a proportionately smaller amount of proteids; it is light straw-coloured, varies immensely in amount (from half a pint to over ten); it is generally free in the pleural cavities, but may be encapsuled or loculated by previous adhesions.

Symptoms.—Dropsy of the pleura is usually bilateral, although in cardiac cases it may be unilateral. It is seldom found unless there has been previously noticed dropsy of the extremities or of the subcutaneous connective tissue first of all. If, then, in the course of the disease producing dropsy, a considerable increase of dyspnœa takes place, an examination of the chest will very probably reveal the presence of fluid in both sides of the chest.

The physical signs are much the same as those of pleurisy, not necessarily, however, of exactly the same extent on both sides, the main differences being, that when bilateral, although the diaphragm is depressed, there is no displacement of the mediastinum and its contents, and the upper line of the fluid is invariably straight and not curved. In the event of the disappearance of the dropsy, the lung is able to re-expand well, as in uncomplicated cases there are no adhesions which might militate against such a result. The extent of the dulness is seldom very extensive.

Treatment.—The treatment must be directed towards the disease of which dropsy is a symptom. Aspiration of the chest may become imperatively necessary when the dyspnœa is urgent, but under ordinary circumstances this procedure is not to be advised. It is better to relieve the dropsy of the subcutaneous tissues of the lower extremities by puncture, either by simple pricking, or incision, or by means of small cannulæ which may remain *in situ*, and these, being provided with small indiarubber tubes, will allow the fluid to be collected into a suitable vessel upon the floor. Great care should be taken that the wounds be kept aseptic. It is found that an outlet of the kind mentioned will act as a general drainage, not only of the subcutaneous tissue, but also of the serous cavities.

In suitable cases venesection will tend to considerably relieve the dyspnœa from which the patient suffers, by relieving the venous engorgement and the dilated right heart.

HÆMOTHORAX.

In this condition blood collects in the pleural cavity. As a rule, hæmothorax depends upon traumatic causes, such, for example, as penetrating wounds of the chest, fractures of ribs with laceration of the lung tissue, or severe injuries to the chest as in falls from horseback, buffer accidents, and the like. These are considered to be surgical affections, and are treated of in books on surgery. Hæmothorax may, however, occur from wounds of an intercostal artery in the operation of paracentesis, or from rupture of an aneurysm, or very rarely from rupture of a pulmonary vessel.

Symptoms.—These depend upon its cause. Rupture of an aneurysm is almost immediately fatal, as would also be rupture of a pulmonary vessel. If the hæmorrhage be gradual, pain and dyspnœa may be present with anæmia and partial collapse. If not immediately fatal, pleurisy may follow the bleeding.

The physical signs are those of fluid in the chest, and have been treated of under the head of Pleural Effusion, but their character would depend upon the amount of the hæmorrhage and the elasticity of the chest walls, as well as upon the movability of the mediastinum and its contents.

When the affection is not immediately fatal, unless it depends upon a condition only temporarily improved, as when a leaking from an aneurysm is stopped, the prognosis is favourable to the extent that absorption of the blood is very probable.

Treatment.—The main object in treatment at first is to stop the bleeding, and the usual methods for so doing may be tried. Absolute rest is necessary, and the injection of a small dose of morphine hypodermically is recommended, but the use of ergotin and other hæmostatics is seldom any good. In the majority of cases of traumatic hæmothorax, absorption of the blood takes place speedily and spontaneously, but in some cases the incision of the chest and the removal of clots has been done by army surgeons with good effect.

PNEUMOTHORAX.

Pneumothorax is the condition in which there is air in the pleural cavity. Pure pneumothorax, although possible (occurring in about 10 per cent. of the cases), is rare; it is almost invariably complicated by the presence of fluid. The fluid is usually pus (pyopneumothorax), but sometimes serum (hydropneumothorax), and occasionally blood (hæmatopneumothorax).

Etiology.—Air enters the pleural cavity, either from a wound of the chest wall and parietal pleura, or through an opening in the visceral pleura covering the lung. The pneumothorax may be complete when the cavity is free from adhesions, or incomplete or loculated when the air enters into portions of the cavity encapsuled by adhesions.

The entrance of air into the pleural cavity by perforation of the parietal pleura is most commonly the result of operations to relieve empyema, and as the opening remains free, the passage of air into the cavity is direct and uninterrupted, but the opening may be spontaneous when an empyema points externally. Gunshot wounds or stabs, not often seen in civil practice, would also perforate the parietal layer as would openings by ulceration of the œsophagus or stomach into the pleura. Perforation of the visceral pleura generally occurs when a superficial emphysematous bulla bursts by overstraining, in which case the pneumothorax may be uncomplicated by the presence of fluid in the cavity, but much more commonly takes place by the rupture of a superficial tuberculous vomica, a disaster which may occur early or late in the course of phthisis. Perforation from ulceration or necrosis of the tissue may occur occasionally in cases of gangrene, cancer, or abscess of the lung. Perforation of the visceral pleura may also take place when an empyema causes ulceration, and, perforating the pleura, discharges through the lung. Finally, accidents, such as severe blows, contusions, falls, or excessive pressure, may cause rupture of both layers of the pleura and pneumothorax, by lacerating the lung at the same time; as may also fractures of the ribs, by the sharp and irregular edges being turned inwards. In the traumatic cases, however, in all probability in addition to air, blood will be found in the pleural cavity. Taking all these causes together, we are able to appreciate their relative frequency best. Thus, according to Saussier, in 131 cases of pneumothorax which he collected, no less than eighty-one occurred from rupture of a phthisical lung or vomica; twenty-nine from empyema; seven from gangrene; five from emphysema; three from pulmonary apoplexy; two from hepatic fistula; and one each from hæmatothorax, cancer, abscess, and hydatids. According to other observers, the proportion of cases due to tuberculous disease is still higher. As regards the air or gas in pneumothorax, its composition no doubt varies with the freedom of communication with the external air, but, speaking generally, the chief gases present are carbon-dioxide and nitrogen, the proportion of oxygen being small. The amount of gas in the pleura varies from a few to a couple of thousand cubic centimetres, and depends upon the condition of the thoracic viscera, whether much bound down with adhesions, solidified by tubercle, or other disease; upon the amount of fluid present; the expansibility of the chest walls; the freedom of mobility of the mediastinum and its contents; and finally, upon the nature of the wound of the pleura, whether valvular or not. In the former case, the air enters the pleura in inspiration, and little may pass out on expiration.

Pneumothorax may occur at any age and in either sex, the male, rather more frequently than the female. It is rather more frequent on the left than the right side.

Anatomical appearances.—On post-mortem examination of cases of pneumothorax, the distension of the thoracic cavity may be great or small, as may be also the displacement of organs; sometimes a greater displacement is present than if the pleura had been filled with fluid only; in others, in consequence of the mediastinum and its contents being bound down by previous adhesions, the displacement may be slight or inappreciable; and the same applies to the condition of the diaphragm and the abdominal organs. Similarly, the lung may be found quite collapsed or but little displaced, according to its condition, whether compressible or solid or nearly so. The fluid found is generally pus, but it may be only slightly purulent and occasionally serous. The amount of the fluid varies from 50 to 1000 c.c. The opening may be large and patent, or may be small, valvular, and difficult to find, unless the lung be placed in water.

Symptoms.—In speaking of the symptoms of pneumothorax, we have to deal only with those cases in which there is no external opening into the pleura. We have already dealt with discharging empyema, or what is sometimes called fistulous empyema.

The symptoms commonly present are pain in the affected side and dyspnœa, with a greater or lesser amount of collapse. In some cases, however, not one of these signs of perforation of the pleura is present, and the pneumothorax can only be detected by physical examination of the chest. The pain in the side may, however, be sudden, sharp, and severe, and the patient may feel as though something had given way in his chest. It is usually felt at the situation of the lower ribs. Dyspnœa may be of a marked type, and may come on suddenly, or (as is most likely) it may be an accession of previously existing difficulty of breathing. The patient may be unable to lie on the affected side, and nearly always has to be propped up in bed. There may be added to this, cough with more or less blood-stained expectoration. The collapse may be very severe, and may almost at once terminate in death, if the patient is weak and exhausted by previous illness. The lips, tongue, and extremities may be cyanosed. The pulse is small and weak. These symptoms may be followed, if the patient survive, by subacute fever.

On physical examination of the chest, the whole of the affected side may be seen to be more or less bulged with pushing of the interspaces outwards. The side does not move on inspiration, and the cardiac impulse may be displaced away from the affected side. On measurement of the chest, the affected side may be found as much as three inches in excess of the other in circumference. On palpation, the vocal fremitus may be diminished or abolished, and, when there is much distension of the affected side, the liver or spleen may be displaced downwards. The heart-beat, too, may be felt in its new position.

On percussion, the resonance over the pneumothorax is tympanitic above, and dull over the fluid at the base of the pleural cavity. The tympanitic note may be elicited as far as the edge of the sternum away from the affected side, and the heart dulness may be found on the sound and not on the affected side. On auscultation, the breathing sounds are feeble or absent if the lung is collapsed, except behind in the vertebral groove. If, however, there has been solidification of the lung, previous to the pneumothorax, there may be bronchial or tubular breathing, or the sounds may be accompanied by an amphoric hum. There are also metallic tinkling, metallic whisper and echo, sometimes pectoriloquy. The succussion sound on shaking the patient is marked only if the fluid be present in considerable amount. The most characteristic physical sign, however, is the bell sound on auscultatory percussion with two coins, or, if the sound is not so clear, it may be what is called anvil sound.

When the pneumothorax is localised to parts of the pleural cavity by adhesions, the physical signs naturally depend upon its extent and position. If the fluid is in large proportion to the size of the cavity, the amphoric signs are few, masked, and a bubbling only may be present. If such a pneumothorax communicates freely with a bronchus, the diagnosis from a superficial cavity of the lung is very difficult, especially as there is probably dulness and not hyper-resonance on percussion.

It is seldom that pneumothorax ends in recovery, except in the cases of rupture of an emphysematous lung, otherwise healthy. In such cases the wound heals, and the gas effused into the pleural cavity is absorbed. Generally death, either immediate, within a few hours, or delayed for some days, follows.

Treatment.—The treatment is obviously only palliative and symptomatic. Generally speaking, stimulants, such as hot brandy and water or ether, should be given, and if the first shock is recovered from, some opiate should be administered. It is best either to put on a linseed meal poultice over the side to relieve pain, or to strap the side. If the dyspnœa be very urgent, and the dislocation of the mediastinum from the pressure of air causes it, puncture of the side with a fine trochar is necessary, and temporarily relieves the patient. This operation may be repeated if occasion requires it.

If the patient recover from the first shock, and survive the first few hours, he should be kept absolutely quiet in a well-ventilated and warm room, and should be fed with very nourishing foods, such as milk, chicken-broth, and strong beef-tea, and some stimulant, such as champagne, brandy, or whisky, should be regularly given. Opiates are very important in this after-treatment. It may also become necessary to puncture the chest to remove the fluid, and if the wound remain open the dyspnœa from the re-accumulation of air is of course removed.

TUBERCLE.

Tuberculosis of the pleura is invariably associated with tubercle of other parts, and can scarcely be considered separately. It may be part of an acute general tuberculosis, in which case small grey transparent tubercles are found in the substance of the pleura, with tubercle in the lungs and other organs. In such cases, symptoms connected with the pleura may or may not have been present. Tubercle is, however, much more common when secondary to tubercle of the lungs. Under such circumstances the new growth is most likely to appear in the immediate locality of the pulmonary deposit. It may also arise in the new membrane which results from previous inflammation. It is said that tubercle may appear first of all in the pleura in cases of old people, and probably in connection with the same affection of the alimentary canal or abdominal organs: as far, however, as our experience goes, this connection, must be very rare.

Sero-fibrinous pleurisy is, as before stated, very apt to be the result of tubercle of the pleura, or of the subjacent scanty subpleural tissue, and may, as we have mentioned elsewhere, be attended with continued fever of an obstinate type, very slow absorption of the effused fluid, and, in some cases, by hæmorrhage into the pleural cavity of a greater or lesser amount, from giving way of the new-formed vessels in the pleural deposit.

NEW GROWTHS.

Malignant new growths of the pleura can hardly be separated, by any symptoms or physical signs which they produce, from similar growths in the lung and mediastinum, to which they are nearly always secondary. In some cases, however, new growths encroach upon the pleura by penetration from outside, and rarely the pleural new growth is the first secondary deposit in the chest. The usual course, however, is for the growth in the lung to impinge upon and implicate the pleura, growing out from it in nodular masses of greater or lesser size. When in this situation, from the position they assume, they become flattened, projecting but slightly, or may be round and prominent. Large growths may grow backwards and compress the lung, or may increase outwards and press out the ribs and sternum. They may also displace the heart and mediastinum. Pleural transudation or actual effusion may result from and complicate the signs of the pleural new growth. For the diagnosis of this form of disease, reference should be made to new growths of the lungs, from which it is practically inseparable.

V. D. HARRIS.

DISEASES OF THE MEDIASTINUM.

By the term mediastinum we denote the space in the middle of the chest between the two pleuræ. By anatomists this space has been divided into four parts, namely, anterior, middle, posterior, and superior. The anterior mediastinum is the part between the sternum in front and the pericardium behind. As the pleuræ come together above behind the second bone of the sternum, the space may be said to be bounded in front by the sternum, and the costal cartilages of the fifth, sixth, and seventh ribs and by the triangularis sterni muscle. The space contains small (anterior mediastinal) lymphatic glands. The middle mediastinum contains the heart and pericardium, ascending arch of the aorta, the main pulmonary artery, and the lower half of the superior vena cava, the phrenic nerves, the arch of the azygos vein, and the roots of the lungs, and the bronchial lymphatic glands. The posterior mediastinum is the space between the pericardium and roots of the lungs, and the spinal column from the fourth dorsal vertebræ downwards. It contains within it the descending arch of the aorta and the descending thoracic aorta, the vagi, œsophagus, thoracic duct, and posterior mediastinal lymphatic glands.

The superior mediastinum is bounded below by a plane drawn from the body of the fourth dorsal vertebræ, behind to the junction of the manubrium with the body of the sternum in front. The upper limit is the upper opening of the chest. It is bounded in front by the manubrium sterni and the origins of the sterno-hyoid and sterno-thyroid muscles, and behind by the spinal column and the lower end of the longus colli muscles. It contains the trachea, œsophagus, and thoracic duct; the transverse arch of the aorta, and the vessels branching off from it; the innominate veins and superior vena cava; the phrenic, vagi, left recurrent, laryngeal, and cardiae nerves; the cardiac lymphatic glands, and the remains of the thymus gland.

In the descriptions given by pathologists, the superior mediastinum is invariably considered as part of the anterior mediastinum.

NEW GROWTHS.

New growths of the mediastinum are comparatively rare affections, one case occurring in about every 150 of diseases of the respiratory system. Malignant growths are much more common than non-malignant, and they are more often primary than secondary. Very little can be said as to the causation; blows on the sternum, or other injury, catching cold, and hereditary predisposition have been mentioned in this connection, without much basis in fact; but observers are generally agreed that males are more liable to the disease than females in proportion of two or more to one. The age most liable seems to be from 30 to 50—37 years of age in males and 36 in females in carcinomatous cases, and 30 to 35 in males, and 35 to 40 in females in sarcomatous cases.

Pathology.—Of the various forms of growth, carcinomata and sarcomata are by far the most common; but opinions are divided as to which of these growths is more frequent, Hare giving 134 of carcinoma to ninety-eight of sarcomata, whilst in seventeen eases collected by ourselves recently, in which the diagnosis was verified by microscopic examination, thirteen were cases of sarcomata. Of carcinomata the most common form is soft or encephaloid, then scirrhus, rarely epithelioma and colloid cancer. The growths may be primary or secondary, more often the former. Of sarcomata the almost invariable form is round-celled or lympho-sarcoma, and the disease is nearly always primary in the mediastinum. Of the other forms of new growth, pure lymphadenomata, fibromata, lipomata, and osteomata occasionally occur, and dermoid cysts have been known in the same situation. The growths begin more frequently in the anterior than in the posterior mediastinum, sometimes in both, only very seldom in the middle mediastinum. The size may vary enormously, in exaggerated cases filling up the whole of one side of the chest. The origin of the malignant growths is probably in the lymphatic glands in some part of the mediastinum; sometimes, according to Virchow, in a persisting thymus. Other origins have, however, been suggested, namely, the fat and connective tissue of the mediastinum, the pericardial connective tissue, the periosteum of the sternum, or the adventitia of the vessels. The direction in which the growths spread is probably most often backwards, involving and pressing upon the heart, and lung of one or other side or of both sides. Sometimes in its spread the growth involves the great veins and nerves, obliterating some by prolonged pressure. Occasionally, the growth may grow upwards and may appear in the neck, involving both arteries and veins. When in the posterior mediastinum, the aorta, the œsophagus, or the spinal column and its contents may be involved. The diaphragm may be pressed down, and the form and size of the chest may be altered by the growth of the tumour outwards, with, or without erosion of the ribs or sternum. From pressure upon blood vessels, there may be dilatation of the external veins, œdema, thrombus, more rarely ascites and swelling of the extremities.

When the growth appears as secondary to malignant disease elsewhere, it more often begins in the posterior mediastinum. No rule can be laid down as to the seat of the primary disease when it is secondary in the mediastinum. As regards sarcomata, however, Hare says that under such circumstances the seat may be the pleura. Extensions or metastases of the disease may occur in the lung, liver, spleen, kidneys, or mammæ.

Symptoms.—The diagnosis of a commencing growth in the mediastinum is exceedingly difficult, if not impossible, as at first there may be no symptoms directing attention to the condition, and even, as the growth becomes larger and larger, the main reliance must be placed upon physical examination of the chest. At some time or other in the course of the disease there is probably pain of some kind, increased on exertion, either over the sternum or elsewhere, occasionally far from the seat of the disease; occasionally tenderness on pressure. Difficulty in breathing is always present late in the case, and may occur quite early. Sometimes the inspiration is accompanied by a piping sound. Cough is a fairly constant symptom; it is still more diagnostic, when it is accompanied by blood-stained viscid expectoration. Hæmoptysis occasionally is present, and has been known to cause death. Of other symptoms more or less common may be mentioned alteration in the voice, which may be whispering, harsh, coarse, or stridulous; headache, vertigo, tinnitus aurium, pains in the throat or dysphagia, emaciation which gradually increases; inequality of the pulses occasionally occur. The presence of tumours elsewhere will materially aid the diagnosis.

Physical signs.—On inspection, in advanced cases, at all events, the veins over the neck, chest, and abdomen are seen to be more or less enlarged and tortuous. There may be general enlargement of one side, or bulgings of the chest here and there, or fulness above one or both clavicles. Sometimes the sternum is pushed forwards. The movement of the ribs on one or both sides is diminished, and the apex beat of the heart may be displaced. On palpation the vocal fremitus is diminished or lost over the site of the growth. On percussion there is generally absolute dulness and increased resistance. The dulness is irregular in shape and size, and merges into the normal præcordial dulness. The dulness is not altered with the position of the patient. On auscultation the vocal resonance is diminished or lost, and the breathing sounds over the situation of the dulness may be absent, or may be harsh vesicular, bronchial, or even amphoric.

All of these physical signs may be modified by the existence of complications, such as pericarditis, pleurisy with effusion, and pneumonia, as also by the blocking up of one or other bronchus, and by pressure upon the trachea.

Course.—Generally speaking, the onset of mediastinal growth disease is insidious and slow; but occasionally symptoms of fever and those of pleurisy may appear at the beginning of the affection. In rare cases, according to Risdon Bennett, there may be hectic fever throughout the whole course of the disease, which does much to confuse the diagnosis. According to our experience of eighteen cases, the duration of the disease was on an average about six months.

Complications.—Several of these have been incidentally mentioned. The most common are pleurisy with effusion, which may be but is not generally purulent, or hydrothorax from pressure on the azygos vein. Pericarditis with blood-stained effusion of a severe type is sometimes present, and a low form of pneumonia, which may terminate in gangrene.

Diagnosis.—The diagnosis is in the early stages exceedingly difficult, but as the growth increases it becomes less so. The most likely conditions to be mistaken for a new growth in the mediastinum are: chronic pleurisy, with great thickening of the chest wall; pleurisy with effusion; aneurysms of the aorta; tuberculosis of the lung; pericardial effusion; mediastinal abscess.

From pleurisy with effusion or with great thickening of the chest wall.—According to our experience, the diagnosis from pleurisy is perhaps the most difficult of all the above-mentioned conditions. When, however, the effusion in pleurisy extends beyond the midsternum, the displacement of the apex beat of the heart is probably greater than would be the case with mediastinal growth. The dulness in the latter case generally begins in front and spreads upwards rather than downwards, except when the growth is in the posterior mediastinum. In chronic pleurisy, with thickening, there is probably contraction rather than bulging of the chest walls, and the heart is, if displaced at all, displaced towards the affected side, and the diaphragm pulled upwards in the same direction, so that the stomachic resonance begins above its usual position. When pleural effusion complicates mediastinal growth, the diagnosis is almost impossible from physical signs alone, and is only arrived at after paracentesis. The onset of the disease is, however, generally different to that of pleurisy, and, as we have seen, the presence of fever is not common.

Aortic aneurysm.—The percussion dulness over an aortic aneurysm is usually situated over the manubrium sterni, and to the right below this, is more definite in shape, and increases very slowly, if at all, for weeks at a time; the percussion resistance is less, and there is probably pulsation in the situation of the dulness; the heart sounds over the dulness are increased, or the second sound is, at any rate; there may be a single or double murmur. The pulses are more likely to be unequal, as are also the pupils, and laryngeal stridor from implication of the recurrent laryngeal is more probable. All of these signs may, however, be present in mediastinal growths, and in both kinds of disease there may be also venous dilatation and œdema of the chest wall. In doubtful cases a skiagram of the chest may be found useful.

Tuberculosis of the lung.—The position of the dulness and the direction of its increase in phthisis, and the presence of tubercle bacilli in the sputum, together with the crepitations heard over the position of the solidified parts, would be, as a general rule, sufficient to point the diagnosis. When tubercle in the lung complicates the course of new growth in the mediastinum, the diagnosis becomes of extreme difficulty, unless the areas of dulness are distinct and do not coalesce.

Pericardial effusion.—When pericardial effusion is well marked, the shape of the præcordial dulness should be pyramidal, with the apex upwards. There is usually a friction murmur to begin with, and the pain, dyspnœa, and cyanosis are much more acute when the pericardium is inflamed primarily. When pericarditis complicates a mediastinal growth, the diagnosis becomes more difficult, but in that case the dulness has been present before the symptoms of pericarditis develop themselves, and should suggest a local cause for the inflammation of the serous membrane.

Mediastinal inflammation and abscess, or mediastinal cyst, will be dealt with in a subsequent section.

Treatment is wholly palliative. Cough and pain may be relieved by opiates, or by counter-irritants such as blisters, over the situation of pain. Venesection sometimes temporarily diminishes the venous obstruction. Tracheotomy has sometimes to be performed to remove immediate danger of death. In some instances, it is of temporary use to remove fluid from the pleura by aspiration.

INFLAMMATION AND ABSCESS.

Inflammation of the mediastinum is a rare affection. It is sometimes simple, ending in recovery or in slight fibrous thickening; or it may be suppurative, and terminate in abscess. Of the latter variety, H. A. Hare has collected the histories of one hundred and fifteen cases, distributed throughout medical literature. Of these, seventy-seven permit of comparison, and fifty-eight are found to belong to the acute variety, and the remainder to the chronic or cold form. The anterior mediastinum is generally the seat of the abscess in the proportion of five to two. Acute abscess is rather more common than chronic; it is, as a rule, traumatic, from stabs or gunshot wounds, sometimes coming on in the course of the acute exanthemata, or from extension of inflammation in the neighbourhood, whereas the chronic appears to be connected with inflamed tuberculous glands. Males are much more often affected than females, in the proportion of fifty-eight to seven. The most likely age appears to be from 28 to 30.

Thomas Harris has called attention to what is called indurative mediastino-pericarditis, in which there is increase of the fibrous tissue of the anterior mediastinum, with or without adherent pericardium. The disease is rare, and mostly affects males, 17 out of 22 cases.

Symptoms.—The chief symptom which distinguishes abscess of the mediastinum from other affections of the same locality is pain; this is, of course, the more intense, if the inflammation producing it is acute. The pain is situated over the sternum or costal cartilages, or may radiate and be most marked at some distance, *e.g.* at one or other shoulder; the pain is boring, lancinating, or pulsating, or only dull and aching. It is generally constant, and is accompanied by more or less marked fever, which may come on at the commencement of the affection with a rigor. When there is marked effusion, these symptoms are accompanied by those of pressure upon neighbouring organs, vessels, and nerves, such as we have seen may accompany mediastinal growth, *e.g.* dyspnœa or orthopnœa, syncope, cyanosis, and œdema, with dilatations of superficial veins, irregularity of the heart beat, cough, and occasionally dysphagia. In acute abscess there is a tendency to point in the neighbourhood of the costal cartilages, or above the sternum, but the pus may cause erosion of the sternum or tract into the abdomen.

The physical signs are practically the same as those of a growth as regards the dulness, which, however, is said to alter on the movement of the patient. The diagnostic sign, however, is the gradual appearance of a fluctuating tumour at one or other border of the sternum, or in the episternal notch. It may pulsate, and in such respect resemble an aneurysm, but the pulsation is not expansile, and the other signs of aneurysm are absent, as there is no alteration of the loudness of the heart sounds or cardiac bruit.

Diagnosis and prognosis.—This is aided by the fact that the symptoms and signs follow upon some injury in the great majority of cases. The duration of acute abscess varies from a few days to as many months, but a chronic abscess may go on for years. As to the indurative mediastino-pericarditis, the symptoms are chiefly those which occur in adherent pericardium and hypertrophy and dilatation of the heart, viz. dyspnœa, cyanosis, and dropsy. The prognosis is serious, since death appears to follow all cases of acute abscess in which the pus does not discharge externally either spontaneously or by the aid of the surgeon.

Treatment.—Possibly the application of leeches or of counter-irritation may in some cases prevent the formation of pus, but if pus has formed, and the diagnosis is certain, the only treatment appears to be to let it out, if operation for that purpose is possible. In some cases it appears that the balance of surgical opinion is in favour of trephining the sternum.

OTHER AFFECTIONS.

Of the still rarer affections of the mediastinum may be mentioned the following:—

Hydatid cyst.—This affection is exceedingly rare, occurring in this situation once in every seventy times. If the diagnosis is certain, endeavour should be made, if possible, to evacuate the fluid.

Simple cyst.—Simple cysts are still more uncommon, but may occur.

Gummata, which are mostly situated in the connective tissue.

Emphysema.—Generally in connection with the operation of tracheotomy, or with pneumothorax.

Persistent thymus gland or enlargement of the thymus may give rise to the symptoms of a new growth in the superior mediastinum.

Enlarged mediastinal glands, either from inflammation (simple or suppurative lymphadenitis), or from the presence of tubercle. In the bronchitis of measles, and in simple broncho-pneumonia, the bronchial glands may be greatly enlarged and infiltrated, and the tissue about them œdematous. Abscess in the bronchial and tracheal glands is occasionally met with. If the bronchial glands are enlarged, and the patient throws back his head, on the application of the stethoscope to the upper border of the sternum, a purring respiratory murmur is heard. The diagnosis of enlarged glands in any other part of the mediastinum is little helped by physical examination.

V. D. HARRIS.

SECTION VIII.

DISEASES OF THE KIDNEY.

INTRODUCTORY.

VARIATIONS IN THE URINE.

The changes that the urine undergoes in disease are dependent either on morbid processes in the kidney, affecting directly the composition of the excreted fluid, or else on morbid processes due to disordered metabolism in other parts of the body, indirectly affecting the composition of the urine. In disease the proportion of the normal constituents of the urine may undergo variation, or else entirely abnormal substances may be present.

Variations in the normal constituents.—**Urinary water.**—The quantity of urine excreted by the healthy adult varies from 40 to 50 oz., the extreme limits probably being a minimum of 20 and a maximum of 80. In health, the quantity of water excreted by the kidney is determined by (1) the amount of fluid consumed, and (2) the amount of fluid eliminated by other channels, more especially the skin, the lungs, and the bowel. In disease a further factor is introduced, owing to the amount of water present in the tissues undergoing considerable variation; whereas in health the quantity in the tissues remains fairly constant. All forms of dropsy will tend to diminish the quantity of urine, whether the dropsy be of renal or of cardiac origin. In health and in disease the quantity of water eliminated by the kidney is ultimately determined by the functional activity of the glomerular tuft and the glomerular epithelium, and the functional activity of these structures is greatly influenced by the rate of the blood flow through the renal vessels. The condition of the renal vessels is the best known factor influencing the rate of secretion of the urine. The variations in the condition of the renal vessels are usually brought about either by causes acting through the vasomotor system, or by substances affecting directly the muscular coat of the blood vessels. It is possible that the rate of the urinary secretion may be influenced through the nervous system, apart from any direct effect produced on the renal blood vessels, inasmuch as complete suppression of urine may occur as a result of the reflex excitation of various parts of the body, without any profound effect being produced on the renal circulation. Further, there is experimental evidence that diminution in the quantity of renal substance, produced by removal of the whole of one kidney and a portion of the other, will in some way influence the quantity of urine, as under these circumstances the quantity secreted is greatly increased.

In disease the quantity of the urine may be increased or diminished, and the increase or diminution may be temporary or permanent. Diabetes mellitus and diabetes insipidus produce a permanent increase in the renal flow, and an increase considerable in amount is usually seen in lardaceous disease of the kidney and in renal cirrhosis, although in the former the increase is only seen in certain stages of the disease. In chronic Bright's disease the quantity may be either increased or diminished. Where dropsy is present the quantity is diminished, but in the chronic contracted white kidney the flow of urine is usually increased. In acute Bright's disease the quantity is greatly diminished. Diminution in the quantity of urine is sometimes so extreme as to cause suppression, and two varieties of suppression are usually recognised, so-called obstructive and non-obstructive suppression. In obstructive suppression a mechanical impediment to the exit of the urine is present, either in the renal pelvis or in some portion of the ureter. In non-obstructive suppression no such impediment is found, and the condition is associated with acute inflammatory and other destructive diseases of the kidney, or in some cases as a terminal phenomenon in chronic destructive diseases. In a few cases non-obstructive suppression is seen to occur with more or less healthy kidneys, as a result of reflex effects produced by morbid processes in other parts of the body. Perforative peritonitis may sometimes lead to complete suppression, and this may also occur after injuries, and even after such an apparently trivial operation as the passage of a catheter. In the latter case it is, however, more usual for suppression to occur where the kidneys are more or less diseased, as in cases of pyelonephritis.

In cases of complete obstructive suppression, life is usually prolonged for a more considerable period than in cases of non-obstructive suppression: and in obstructive suppression very few symptoms are produced, except progressive weakness and a fall of body temperature, with contraction of the pupils. To this condition the term of latent uræmia may be applied. In non-obstructive suppression severe uræmic phenomena of the usual type are generally produced. In some cases, however, of non-obstructive suppression, even when associated with complete destruction of the kidney, latent uræmia, and not acute uræmia, is seen.

Specific gravity.—The specific gravity of the normal urine varies from 1015 to 1025. In disease it may fall as low as 1002 or 1003, as in diabetes insipidus, or it may rise as high as 1060. In health, after copious drinking, the specific gravity may fall, temporarily, to below 1005. In observations on the specific gravity of the urine it is essential that the specimens observed should be samples of the whole twenty-four hours' secretion, as the influence of meals and of drinking on the specific gravity is so great as to cause very serious errors, unless this precaution be taken. A persistently low specific gravity, *i.e.* below 1010, may usually be taken as evidence of the existence of disease, and more especially of such conditions as the granular kidney, the so-called surgical kidney, the cystic kidney, and lardaceous disease. In chronic Bright's disease the specific gravity is usually low, but cases are seen exceptionally where the specific gravity, even in fatal cases, remains high. A specific gravity above 1030, more especially above 1035, is very suggestive of the presence of sugar; but very dense urines, containing large quantities of urea, but no sugar, are occasionally met with, having a specific gravity of 1040 or more. Sugar may be present, however, in the urine, with a specific gravity as low as 1010; and even where sugar is present, variations in the amount of sugar do not necessarily produce corresponding variations in the specific gravity; a urine with a higher specific gravity may contain a less percentage of sugar than another urine with a lower specific gravity. This arises from the fact that other constituents of the urine besides sugar materially influence the specific gravity. If the urine be very abundant, as in the more severe forms of diabetes, then the variations in the specific gravity and in the amounts of sugar present may more or less coincide, as in such cases the specific gravity is almost entirely dependent on the quantities of sugar present.

Reaction.—The urine normally is acid, owing to the presence of acid phosphate of soda, and the acidity of the total urine of twenty-four hours is equivalent to 2 grms. of oxalic acid. The normal healthy urine is most acid in the morning, inasmuch as the influence of meals during the day is such as to cause the so-called "alkaline tide." Urine secreted two to three hours after a meal may be alkaline, but more usually its acidity is only diminished. This diminution in the acidity of the urine is dependent mainly on the large quantity of alkaline salts ingested with the meal, more especially with vegetable food. The urine actually secreted two to three hours after a meal is probably alkaline, but when voided it is still acid, owing to the fact that it becomes mixed in the bladder with acid urine; hence it is more usual for the acidity of the urine simply to be diminished by meals. The reaction of the urine is profoundly influenced by diet, being more acid with a meat diet, and more alkaline with a vegetable diet. This is chiefly dependent on the presence of acid phosphate of soda in meat. In disease the acid reaction of the urine may be either increased or diminished. Increase is especially seen in cases where a highly concentrated urine is secreted, as in febrile diseases, and in cases where large quantities of meat are eaten, also in the condition of so-called lithæmia. In diabetes, and more especially in diabetic coma, the urine may be highly acid, from the presence of β-oxybutyric acid; and in cirrhosis and other destructive diseases of the liver the acidity may also be increased, and lactic acid may be present. In other diseases the acidity of the urine may be diminished, and this is especially the case in dilated stomach. The urine may become alkaline, and two varieties of alkaline urine are recognised, one where it is dependent on the presence of a fixed alkali, and the other where it is due to a volatile alkali. Alkalinity from a fixed alkali may be present as a result of vegetable diet, but it is more especially seen in cases of dyspepsia and neurasthenia; and in such cases the mechanism of its production is obscure. It is not of any great importance, except that it may cause the urine to become milky from the precipitation of amorphous tricalcic phosphate. In exceptional cases this may lead to the formation of stones. Alkalinity from a volatile alkali is a much more serious condition, and is dependent on the decomposition of the urine in some portion of the urinary channels, with the formation of carbonate of ammonia. This result is generally secondary to infection of the urine with micro-organisms, more especially by the introduction of infected catheters. In exceptional cases the organisms causing the decomposition of the urine reach the urinary channels from within, as for instance by the rupture of an abscess into some portion of the urinary tract; and occasionally, perhaps, it is possible that micro-organisms may reach the urinary bladder by passing up the urethra, or even by being excreted through the kidney from the blood.

Urea.—Approximately 15 grms. of nitrogen are excreted per diem in health, and the most important nitrogenous constituents of the urine are urea, uric acid, and creatinin; xanthin, guanin, and hippuric acid are also present, but are of no great importance. The daily quantity of urea excreted varies between 20 and 40 grms. The average is usually stated to be 30 grms.; approximately 0·5 grms. of urea are excreted per kilo of body weight. About 2 per cent. of urea is present in the normal healthy fluid, but in health it may rise to as much as 3 per cent. The amount excreted is largely influenced by the diet, and it is greatly increased after meals. Exercise does not cause any appreciable increase, but copious water drinking increases the amount temporarily. Observations on the excretion of urea in disease are of little value, unless the amount and nature of the food taken are considered. In disease, the quantity excreted may be increased or diminished, and the increase may be absolute or may be relative. In the former, the quantities passed are really absolutely increased. Where a relative increase only is present, the quantities excreted are excessive when the diet ingested is considered. Thus the excretion of 30 grms. in the twenty-four hours, by a sick man lying in bed and taking a low diet, compared to that consumed in health, is really an excessive excretion, and must be dependent in part on tissue waste. In diabetes mellitus the quantity excreted is greatly in excess of the normal, and this is mainly dependent upon the increased diet. In some cases, however, it is also dependent on increased tissue waste. In diabetes insipidus the quantity may also be slightly increased. In febrile diseases the percentage of urea is usually increased, and the total amount is always relatively increased, although the actual amount secreted may not be greatly above the normal, and in many cases is below the normal for a healthy adult; but in such cases, owing to the diminished ingesta, there may be really a relative increase. In most wasting diseases the amount of urea is diminished, owing to the great diminution in the appetite, and to the fact that in almost all wasting diseases, owing to the digestive disturbances present, there is usually a condition of partial starvation. In diseases destroying the liver substance, as in atrophic cirrhosis, and more especially in acute yellow atrophy of the liver, there is a considerable diminution in the quantity of urea, and in some cases it may even completely disappear. In renal diseases the excretion of urea may be increased or diminished. In most it is generally diminished, as in acute nephritis, consecutive nephritis, some forms of chronic Bright's disease, and cystic kidneys. This is especially the case in those forms of chronic Bright's disease which are associated with dropsy, and where the quantity of urine passed is greatly below the normal. In the granular kidney, and in other forms of chronic nephritis not accompanied by dropsy, the quantities of urea excreted may be quite comparable to those seen in health. In many forms of renal disease the diminution in the urea excretion is not to be taken as an indication of the inefficiency of the kidney to act as a filter, the real cause of the diminution being the small quantity of food taken, and the still smaller quantity absorbed, owing to the presence of nausea, vomiting, and diarrhœa. The onset of uræmia in chronic renal disease, and in acute renal disease, is often accompanied by a considerable diminution in the quantity of urea excreted, but this is by no means invariable; and acute uræmia in chronic renal disease may be seen, accompanied with the excretion of quantities of urea quite comparable to, or even greater than, those seen in health. Estimations of urea for clinical purposes must be made from samples obtained from the whole twenty-four hours' urine, and the hypobromite method is usually employed for the purpose. This method, when carried out with great care, introduces an error of some 8 per cent., unless sugar be present in the urine. In the latter case, the whole of the urea is decomposed by the hypobromite. Hypobromite of soda decomposes other nitrogenous constituents of the urine besides urea. In performing the test, it is essential that the hypobromite of sodium should be freshly prepared, and that it should not be present in too great an excess; the mixing of the urine with the hypobromite solution should also be carried out very slowly, and the mixture kept cool.

Uric acid and urates.—Uric acid is present in the urine generally to the amount of 1 grm. Occasionally as much as 2 grms. may be excreted in the twenty-four hours. Uric acid is the most important representative in the urine of the group of bodies containing alloxur bases. Uric acid is very insoluble in water, 1 grm. requiring about 15,000 c.c. to dissolve it. In the urine, however, this quantity of uric acid is kept in solution by one-tenth of this quantity of fluid. All observers are agreed that it is not present in the urine, as such. Two main views have been held—(1) that it is present as an acid urate or biurate; (2) that it is in the form of what has been described as a quadriurate. According to the latter view, the bibasic acid, uric acid, would form three orders of salts—the neutral urates, the bi- or acid urates, and the quadriurates, the last being a compound of a molecule of a biurate with a molecule of free uric acid. The neutral urates are of no physiological or pathological importance, inasmuch as that they do not exist in the body either in health or in disease, and are only formed in the laboratory as a result of heating uric acid in sealed tubes with an alkali. The acid biurates are found in the urine as one of the constituents of the deposit of urates, and also in gouty concretions. The main evidence in support of the existence of a third group of salts, the quadriurates, lies in the fact that the ordinary deposit of urates from the urine contains a larger quantity of uric acid than can be accounted for by the bases present, and in some specimens analysed both by Bence Jones and by Sir William Roberts the proportion of uric acid present was twice the amount required to form an acid urate. Further, the ordinary deposit of urates decomposes under a variety of circumstances, and more especially on the addition of distilled water, liberating free uric acid, which is deposited in the well-known crystals. Urine, especially alkaline urine, on heating, dissolves freely large quantities of uric acid, and, when cooled, deposits a sediment similar in character to that occurring with the spontaneous deposition of urates, and, like it, liberating crystals of free uric acid as a result of decomposition. The alkaline blood serum will also dissolve uric acid freely, but here on decomposition the biurate is deposited. It is doubtful whether the uric acid can exist in the blood in the form of a quadriurate, inasmuch as most observers are agreed that an acid medium is necessary for the production of this hypothetical salt. The observers who reject the hypothesis of the existence of uric acid in the urine in the form of a quadriurate, explain the liberation of uric acid crystals from the deposit of urates as the result of an interaction between the urates and the phosphates, the acid phosphate of soda taking a molecule of base from a biurate in exchange for a molecule of hydrogen, with the formation of free uric acid and a neutral or alkaline phosphate. This is especially apt to occur when the urine cools, owing to the fact that the solubility of uric acid, as such, in cold urine is far less than in warm, and the mass influence of uric acid in the cold would be lessened, and so allow the acid phosphate to effect the dissociation of the urate present. On the other hand, when the fluid is warmed again, the solution of the uric acid in the warm liquid would increase the mass action of the uric acid, with the formation of the biurate and the reappearance of the acid phosphate. The view that the uric acid exists in the urine in the form of a biurate, and that the decomposition of this, with the liberation of free uric acid, is dependent on the mass action of acid phosphate of soda, receives considerable support from the fact that all deposits of urates contain a certain proportion of phosphates. All urines deposit uric acid sooner or later, provided the fluid is not allowed to undergo putrefaction, and its deposition is influenced mainly by the acidity of the urine, the quantity of other salts present, and also, to a certain extent, by the amount of pigment. Dilute urines deficient in salts and colouring matter frequently deposit uric acid, whereas concentrated highly acid urines, containing perhaps a larger percentage of uric acid or urates, do not, owing to the uric acid being kept in solution by the other salts, and particularly by the phosphates.

The uric acid in the urine has a double origin, being derived in part from the metabolism of alloxur bases contained in the food, and more especially in flesh, and in part from the metabolism of the tissues. Certain articles of diet, such as liver and thymus, greatly increase the amount of uric acid excreted. With a vegetable diet, or one consisting of milk and bread, the uric acid in the urine is derived almost entirely from the metabolism of the tissues. The most delicate test for uric acid is the murexide test. The suspected substance is treated with fuming nitric acid, and warmed; when cold, ammonia is added, and a purple-red colour is produced. The addition of potash instead of ammonia causes a purple-blue instead of a purple-red colour. There are various methods for estimating the quantity of uric acid in the urine, the simplest being that of Heintz. Ten c.c. of hydrochloric acid are added to 200 c.c. of filtered urine, and the mixture is set aside for forty-eight hours in a cool place. After that interval the crystals of uric acid are collected on a weighed filter, washed and dried at 100° C., and weighed. This method gives only a rough indication of the amonnt of urid acid present, since a considerable quantity may be retained in the acid liquor. A more accurate method is that of Hopkins. Fifty grms. of ammonium chloride are added to 100 c.c. of urine. The precipitate is collected after two hours on a filter, and washed with a saturated solution of ammonium chloride. It is then collected in a small beaker by washing the filter paper with hot distilled water, and it is subsequently heated to boiling with an excess of hydrochloric acid. The uric acid separates, is collected and washed on a filter, and ultimately dissolved in a weak solution of sodium carbonate. The solution is made up to 100 c.c. and mixed with 20 c.c. of sulphuric acid, and then titrated with one-twentieth normal potassium permanganate, 1 c.c. of this solution being equivalent to 0·00375 grm. of uric acid.

The amount of uric acid in the urine is diminished during the paroxysmal stage of gout, but with the subsidence of the attack an increased quantity is excreted. In pernicious anæmia and leukæmia, and more especially in splenic leukæmia, the quantity of uric acid in the urine may undergo great increase. In febrile diseases and in ague it may also undergo some increase, and in certain forms of diabetes, known as "gouty diabetes," the quantity excreted is also above the normal. In ordinary diabetes, notwithstanding the large excretion of other nitrogenous constituents, the urine does not, as a rule, tend to deposit large quantities of uric acid.

In health the urates present in the urine remain in solution, not only when the urine is passed, but also subsequently, when it has cooled. In very hot weather, and after excessive sweating, when a concentrated urine is passed, the urates are frequently deposited as the urine cools, forming the well-known lateritious, or uratic, deposit. This deposit, as mentioned above, undergoes decomposition, with the liberation of free uric acid, provided the urine does not decompose; and the time of occurrence of this liberation of free uric acid is of some importance in pathology. Where it occurs after complete cooling of the urine, and some hours after it has been passed, it is of no importance; but where the liberation of free uric acid occurs within a few minutes of the passage of the urine, it may also occur in the urinary passages, and thus form a nucleus for renal calculi or gravel; and the conditions influencing this liberation of free uric acid from the urates are matters of considerable importance. Although the percentage amount of urates and uric acid present in the urine will influence the separation of free uric acid to a certain extent, other conditions of the urine, such as the amount of other saline ingredients present, and the degree of acidity, are of equal if not of greater importance; and it is well known that highly concentrated urines, containing an abundance of urates and other salts, do not readily liberate free uric acid. Copious deposits of urates occur also in febrile illnesses, and more especially towards the subsidence of the disorder, as is well seen in the case of the common feverish cold. In dyspepsia, gastritis, gastric dilatation, hepatic cirrhosis, and in cases of heart disease, where venous congestion is a marked feature, urates are also deposited from the dense high-coloured urines. The uratic deposit sometimes contains crystals of acid sodium and ammonium urate, and this is more especially the case in children. These crystals are frequently very irregular, with spinous processes, and hence may cause a good deal of irritation of the urinary tract; they may, in children, even give rise to suppression. Both uric acid and the urates tend to reduce sulphate of copper, more especially when the urine is boiled for any length of time with Fehling's solution, and in this way dense high-coloured urines may be thought to contain sugar, when the copper reduction is dependent on the uric acid and urates. The reduction is rarely as complete as that seen with sugar, the deposit being usually of a greenish yellow colour, rather than the brick-red which is so characteristic of sugar. In all cases of doubt, the presence of sugar should be verified by the fermentation test. The other nitrogenous constituents of the urine, such as xanthin, hypoxanthin, creatinin, are of no great clinical importance, although the quantities present, especially of creatinin, are greatly increased in wasting diseases, and even normally more creatinin is present than uric acid; but inasmuch as it is a very soluble body, it is of no great importance, except that, like uric acid, it also reduces copper, and therefore when present in excess may be mistaken for sugar, unless the fermentation test be done.

Leucin and tyrosin.—The amido-acids, leucin and tyrosin, are normally formed in the alimentary canal, and are occasionally found in the urine, sometimes in traces, sometimes in large quantities. When large quantities of leucin and tyrosin are present in the urine, the excretion of urea is greatly below the normal, and ammonia and lactic acid are present. This is more especially seen in destructive diseases of the liver, such as acute yellow atrophy, and the fatty degeneration which is seen as a result of phosphorus poisoning. In cases of acute yellow atrophy, the urea may almost totally disappear from the urine, and the amount of leucin and tyrosin present may be sufficiently great for these substances to be spontaneously deposited. In phosphorus poisoning they may also be present in abundance, but here the amount of urea is more usually greatly diminished, but it is not absent. Leucin and tyrosin in smaller quantities are also found in the urine in other conditions, and more especially in atrophic cirrhosis, where necrotic changes in the liver cells, somewhat similar to those seen in acute yellow atrophy and in phosphorus poisoning, are not uncommon. The presence of those bodies in the urine is always indicative of destructive lesions of the liver substance. Tyrôsin occurs in crystals of a greenish colour, which are usually deposited in the form of sheaves and rosettes. Leucin occurs in globular masses. Crystals of calcic phosphate, sodic phosphate, and of the lime and magnesium salts of fatty acids, are also sometimes found in the urine, in the form of sheaves and rosettes, and may be mistaken on casual examination for tyrosin. In all cases the suspected presence of leucin and tyrosin in the urine should be confirmed by the tests for these bodies. Tyrosin, when heated with Millon's reagent, gives a brilliant crimson colour. Piria's reaction is also a valuable test for the presence of tyrosin. The suspected material is warmed with concentrated sulphuric acid; it is then allowed to stand, and diluted with water saturated with barium or calcium carbonate, and filtered while hot. On the addition of dilute ferric chloride to the filtrate, a violet colour is produced.

Cystin and cystinuria.—Cystin (amido-sulpho-lactic acid) is a body containing sulphur, and is, in very rare cases, found in the urine in sufficient amount to form a calculus. It is probable, however, that very small quantities of an allied body are present in the urine normally. Cystin crystallises in flat hexagonal plates, insoluble in acetic acid, soluble in ammonia, and it burns with a bluish green flame on platinum. If heated with caustic potash on a silver coil, a black mark is left on the silver, owing to the formation of sulphate.

Chlorides.—10 to 15 grms. of sodium chloride are excreted daily. The quantity is mainly dependent on the amount of chlorides in the food. The chief point of clinical interest with reference to the chlorides lies in the fact that they are diminished in amount during the height of the fever in pyrexial diseases, and more especially in acute pneumonia, where, during the height of the malady, they may disappear, to reappear during convalescence. It is more common for the chlorides in pneumonia to be diminished only, and for traces to be present for one or two days during the height of the disease; and, according to Hutchison, it is rare for the whole excretion throughout the attack to exceed 2 grms. of chloride of sodium per diem. There is no relation between the height of the fever and the degree of chloride diminution, nor between the latter and the extent of lung involved. The diminution usually lasts for one or two days after the crisis, and is succeeded by a sudden and excessive excretion. During the height of the disease there is a retention of chlorides within the body. The urea, the phosphates, and the sulphates are increased during the height of the disease. The diminution of the chlorides of the urine is a very marked phenomenon in pneumonia, but it occurs also in other acute febrile diseases, and especially in acute rheumatism, and in typhus fever. In malaria, however, the chlorides are increased in amount during the pyrexia, and in pleurisy and in empyema the chloride excretion may remain high. In some cases of gastric dilatation there may be a great diminution in the excretion of chlorides. The lack of excretion of chlorides in pneumonia is not due to any vicarious excretion of these substances in the sweat, bowel, or sputum, and the percentage amount of sodium chloride in the sputum is very low. The diminished excretion is also not dependent on any incapacity of the kidney to excrete chlorides, but it is apparently entirely due to some disorder of metabolism in the pneumonic processes by means of which the chlorides are retained in the tissues. To determine roughly the quantity of chlorides present in the urine, the urine should be acidified with nitric acid, and a few drops of nitrate of silver added, and the precipitate obtained compared with one produced by similar measures in a specimen of normal urine.

Sulphates.—Sulphur is excreted in the urine in three forms—first, in the form of sulphates, the sulphuric acid being combined with the inorganic bases sodium, potassium, and magnesium; secondly, some sulphur is excreted in the form of aromatic sulphates, the organic bases phenol, cresol, indol, or skatol being combined with the sulphuric acid and an inorganic base, such as potash, to form potash salts; thirdly, a small quantity of sulphur is excreted in combination with amides, forming bodies allied to taurin (amido-ethyl-sulphonic acid), and cystin (amido-sulpho-lactic acid). The quantity of sulphur excreted in this third manner is small, and the proportion existing between the simple sulphates and the aromatic sulphates is approximately from 12 to 20 of the former to 1 of the latter. The sulphates of the urine are derived in part from those in the food, and in part from the metabolism of the proteid tissue. The aromatic sulphates are derived in great part from the decomposition of proteid matter in the alimentary canal, and to a certain extent the quantity of aromatic sulphates in the urine may be taken as an indication of the extent of intestinal decomposition and putrefaction. They are therefore greatly increased in amount in all conditions of intestinal obstruction, partial or complete. Their formation is not limited, however, to the alimentary canal, and their excretion is increased in all conditions in which there is decomposition of proteid matter, as for instance in cases of suppuration, empyema, especially the putrid form, pulmonary gangrene, etc. In typhoid fever they are said not to be increased. The excretion of aromatic sulphates is increased by the administration of a considerable number of aromatic bodies, of which phenol and camphor are the most important; and in cases of carbolic acid poisoning the increase in the aromatic sulphates may be so great as to seriously diminish the quantity of ordinary sulphates present in the urine. Indican, the potash salt of indoxyl sulphuric acid, is a normal constituent of the urine; like the aromatic sulphates, it generally undergoes a considerable increase in cases of intestinal putrefaction and of putrid inflammation. Approximately, 2 grms. of sulphuric acid are excreted per diem in normal urine.

Phosphates.—The acidity of the urine is normally dependent on the presence of acid sodium phosphate, and from 2 to 6 grms. of phosphoric acid are excreted in the urine per diem. Two-thirds of the phosphoric acid present is excreted in a soluble form, in combination with such bases as potash and soda. The remainder of the phosphoric acid is united with the bases lime and magnesium, and these are only soluble in acid urine. The neutral and basic phosphates of lime are deposited in faintly acid, neutral, and alkaline urines. Urine which is alkaline from the presence of ammonia contains the triple ammonio-magnesium phosphate, and this is occasionally deposited from urines which are still faintly acid.

The phosphates in the urine are derived in great part from the food, and hence the quantity is considerably increased after meals; and it is not uncommon for some deposition of the earthy phosphates to occur, owing to the diminished acidity of the urine. The amount of deposition of phosphatic material from the urine, however, is no measure of the quantity actually present, since the bulk of the phosphoric acid excreted, both in health and in disease, is in combination with sodium and potash forming a soluble salt; and in all cases, if it is desired to determine the quantity of phosphatic matter present, the usual methods of estimation must be employed, and no reliance should be placed on the mere amount of the deposit. Although the total quantity of phosphatic matter excreted is mainly dependent on the composition of the food, yet in febrile diseases the quantity is at first diminished, and later, during defervescence, it is increased. In certain forms of neurasthenia the quantity is also said to be increased, and in diabetes a large excretion is not uncommonly present; but this is probably dependent on the increased appetite so common in this disease. The term, however, phosphatic diabetes, has been applied to a condition in which, along with neurasthenic symptoms, a considerable quantity of urine containing an excess of phosphates is said to be excreted. The deposition of the earthy and triple phosphates from the urine is the most important fact, from a clinical standpoint. Urine that is faintly acid, neutral, or alkaline, when heated frequently, yields a deposit of earthy phosphates. This deposition of earthy phosphates is probably dependent on a decomposition between a dicalcic phosphate and a monocalcic phosphate, with the formation of tricalcic and monocalcic phosphate, 2 molecules of dicalcic with 1 of monocalcic giving rise to 1 molecule of tri- and 1 molecule of dicalcic phosphate. The tricalcic phosphate, being far less soluble than the di- or monocalcic salt, is precipitated. If the urine is not raised to the actual boiling point, or if the heating be done in a sealed tube, the precipitate produced can be re-dissolved on cooling, and the operation repeated several times in succession. Although the above is the most probable explanation of this well-known phenomenon, it has been suggested that the precipitation of the phosphates on heating is dependent on the raised temperature driving off carbonic acid, thanks to which the phosphates were kept in solution. It has also been stated that the mere heating of the urine is sufficient to decompose the urea present with the formation of ammonia.

The deposition of phosphates in the bladder is dependent more on the reaction of the urine than on the amount of phosphatic matter present. The earthy phosphates tend to be deposited when the reaction of the urine is alkaline from the presence of fixed alkali. Occasionally stellar phosphate, a dicalcic phosphate, is precipitated under these circumstances. This deposit may be seen in the urine of diabetes and in malignant disease. Where the urine is alkaline from the presence of volatile alkali, triple phosphate is commonly deposited, and is a frequent cause of the formation of a calculus, the nucleus, however, of which is usually formed of uric acid or of oxalate of lime. Triple phosphates may also form deposits on the surface of the bladder, or in sacculi in connection with it, or even on growths in the bladder; in fact, in any condition in which cystitis is present with sufficient severity for the urine to be alkaline from the presence of ammonia.

Oxalates.—Oxalic acid is excreted to the extent, approximately, of 20 mgrms., in the form of an oxalate in the normal urine, and the oxalates, like the urates, are normally kept in solution, thanks to the presence of acid phosphate of soda. Oxalates tend to be deposited in the urine in the form of oxalate of lime, which crystallises either in dumb-bells, or in octahedra, or occasionally in ovoid crystals. The crystallisation of oxalate of lime in the urine, like that of uric acid, is often affected or modified by the presence of mucin and other colloidal substances, and it is usually held that bodies of this description may modify the crystalline form considerably. A few scattered crystals of oxalate of lime may occasionally be seen in the urine in health, and more especially after certain articles of diet, such as rhubarb and sorrel. The term oxaluria is applied to a condition in which there is a more or less persistent deposit of oxalate of lime in the urine, and it is not uncommon in these cases for a calculus to be formed. Very little is known as regards the conditions which tend to produce oxaluria, except that in many respects they resemble those which may lead to the formation of the uric acid calculus. Calculi formed of oxalate of lime are exceedingly irregular and spinulose, and are liable to cause very considerable irritation of the urinary passages. In some cases the increased excretion of oxalate of lime is associated with dyspepsia, mental depression, neurasthenia, and hypochondriasis; and some observers have thought that these symptoms are associated with and dependent on the presence of oxaluria.

Normal pigments.—The normal colour of the urine is dependent on the presence of urochrome, an amorphous brown substance insoluble in ether and chloroform, but freely soluble in water, yet yielding no banded spectrum. Urobilin, a second pigment, present in the normal urine, but only in traces, plays a very small part in the production of the normal colour. Urobilin is freely soluble in chloroform, and yields a very definite absorption band in the spectrum at F. In health very small quantities of urobilin are present in the urine, but in several diseases it may undergo a great increase in amount. Normal urine also contains traces of uroerythrin, a pigment that can be extracted from normal urine by means of amylic alcohol. Hæmatoporphyrin is also present in the normal urine in traces.

Abnormal pigments.—In disease the modified colour of the urine may be dependent on the presence of normal pigments in excessive amounts, or upon the presence of abnormal pigments.

Urobilinuria.—There are a number of conditions in which the urine has a totally abnormal colour in disease, from the excretion of urobilin in increased amount. In urobilinuria the urine is of a deep brown colour, resembling that seen where bile, or altered blood pigment, are present. Patients with marked urobilinuria are often distinctly yellow in colour, both as regards the skin and the conjunctivæ. Some observers have thought that the urobilin present in cases of urobilinuria is somewhat different from the urobilin seen in normal urine, but subsequent researches have not substantiated this, and in all probability there is no real difference between so-called normal and so-called pathological urobilin. Urobilinuria is seen, especially after copious internal hæmorrhages, such as ruptured tubal gestation, or any other form of large peritoneal hæmorrhage. It also occurs in pernicious anæmia, paroxysmal hæmoglobinuria, cirrhosis of the liver, and in fact in all diseases in which there is an increased destruction of blood corpuscles, either in extravasated blood, or in the blood vessels. Urobilin, in excessive amount, may be the only pigment present, or it may be associated with the presence of other pigments, as for instance the bile pigments or other decomposition products of hæmoglobin. Urobilinuria is recognised, more especially by the spectroscopic appearances of the urine; the urine containing it in large amounts presents a superficial resemblance to urine containing bile.

Choluria.—In jaundice the bile pigments are present in the urine, the colour of which varies, according to the amount present, from a pale reddish brown to almost black. In all cases where bile is present in the urine, the upper surface of the fluid, on oblique illumination, shows a greenish tinge from the oxidation of bile pigment present. Obstruction of the bile duct invariably leads to the presence of bile pigment in the urine, and many observers hold the view that bile pigments never appear in the urine unless such obstruction is present. On the other hand, some consider that decomposition of blood pigment, either in the form of extravasated blood, or still within the vessels, may give rise to choluria; and bile pigments in the urine have been described in cases of internal hæmorrhage, and in cases of paroxysmal hæmoglobinuria. It is probable, however, that in many of these cases the presence of urobilin in large amounts in the urine has been mistaken for the presence of bile pigment; and it is doubtful whether true bile pigments ever appear in the urine except as a result of some condition leading to obstruction of the bile ducts. Bile pigments are readily recognised in the urine in cases of jaundice, but the bile salts are only detected with difficulty and by special methods. Recognition of the bile pigments is readily effected by the use of Gmelin's nitric acid test, but in using this test the presence of bile pigments should not be asserted unless green is the first colour seen in the play produced, as the urine may contain a number of other pigments which will undergo some change of colour, on the addition of a powerful oxidising agent like nitric acid. The bile pigment may be precipitated from the urine by the addition of milk of lime, and obtained from the precipitate by treating this with chloroform acidified with acetic acid. The bile salts may occasionally be recognised by Pettenkofer's reaction, but more usually the urine must be evaporated to dryness, and the bile salts extracted with alcohol, and obtained from the alcoholic solution by precipitation with ether, and the test applied to the purified salts. The recognition of bile salts in the urine is of no great clinical importance.

Hæmoglobinuria.—The presence of hæmoglobin in the urine, apart from the presence of blood corpuscles, is seen in paroxysmal hæmoglobinuria, and also in a number of conditions where the blood corpuscles undergo disintegration, as after the decomposition of various poisons, such as arseniuretted hydrogen, chlorate of potash, carbolic acid. It is also occasionally seen in Raynaud's disease, in malaria, and in septic infections, as well as in some acute infective processes. In paroxysmal hæmoglobinuria the amount of hæmoglobin excreted in the urine may be very large. In many cases the pigment is excreted, not only in the form of hæmoglobin, but along with this there are decomposition products such as methæmoglobin and hæmatin. Recognition of these substances is best effected by the spectroscope, but Heller's blood test is also applicable. On microscopic examination of the urine no blood corpuscles are found, but a number of droplets of a yellowish colour are often seen, together with casts and crystals of oxalate of lime.

Hæmatoporphyrin.—Hæmatoporphyrin is sometimes excreted in sufficient quantity to cause the urine to acquire a deep red or port-wine colour. This occurs more especially after the administration of certain drugs, and more particularly sulphonal. It has also been observed in rheumatic fever, cirrhosis of the liver, peritonitis, and Addison's disease. The pigment can be isolated from the urine by precipitation with barium chloride and barium hydrate, and can then be extracted with acidified alcohol, and identified by the characteristic bands which it yields on spectroscopic examination.

Melanuria.—Melanin is a rare constituent of the urine, but occasionally a urine of a *café au lait* colour is excreted, which, on exposure to the air or on the addition of nitric acid, becomes black, owing to the chromogen of melanin being converted into melanin. Melanuria is occasionally seen in wasting diseases, but is more especially a feature of melanotic sarcoma, although it is not an invariable accompaniment of this condition. Ferric chloride, when added to urine containing melanin, causes a greyish brown or black precipitate, soluble in excess, and bromine water gives a yellow precipitate which gradually blackens.

Alkaptonuria.—In rare instances urine is excreted which, although of normal appearance when passed, acquires a deep brown colour, or even becomes black on exposure to air. The brown colour is intensified by alkalies, and these urines reduce Fehling's solution, so that the error may be made of thinking that sugar is present, but such urine has no rotatory action on polarised light. In many cases this condition is congenital, and in some instances several members of a family have been affected. According to Garrod, in all cases of alkaptonuria, homogentisinic acid is present. The condition is one only of scientific interest, as it in no way causes any ill effects.

Pyrocatechin is present in the normal urine in small amounts, and is greatly increased in cases of carboluria, where hydroquinone is also present. Both these substances are present in combination with sulphuric acid, and urine containing them may be colourless when passed, but darkens on exposure to air, and may become black. These substances are the cause of the greenish black colour of the urine in carboluria.

Indicanuria.—Indigo, in the form of its precursor indican, is present in the normal urine to the extent of approximately 20 mgrms. Indican, the potash salt of indoxyl sulphuric acid, owes its origin mainly to the indol formed in the intestine as a result of proteid decomposition. Indoxyl is also excreted in combination with glycuronic acid. In disease the excretion of indican may be increased to such an extent that in the decomposed urine it may be sufficiently abundant to impart a blue colour to the liquid, and in very rare instances it has been excreted in sufficient amount to form a calculus. Increased excretion of indican is mainly indicative of increased intestinal putrefaction, and hence it is especially marked in long-continued chronic obstruction of the large intestine. It has, however, also been described in cases of putrid inflammation, more especially in putrid empyema and gangrene of the lung. It has also been described occasionally in tuberculous peritonitis, cancer, and gastric ulcer. Indigo can be obtained from the urine by treating it with an equal volume of hydrochloric acid, and adding cautiously, drop by drop, a solution of calcium hypochlorite. On shaking the urine with chloroform, the indigo is dissolved by the latter.

Skatol.—In cases of constipation and intestinal putrefaction, the amounts of potassic skatoxyl sulphate in the urine are considerably increased, and on treating such a urine with a mineral acid the fluid becomes of a deep red colour. The presence of this substance in increased amount in the urine has the same significance as indicanuria.

Albuminuria.—The proteids present in the urine in albuminuria are usually serum albumin and serum globulin. Albumoses may be present in small quantity; peptone, fibrin, and nucleo-albumins are rarer forms, occasionally present.

Albuminuria not dependent on obvious disease.—Normally the urine contains no proteid matter, but the term "physiological albuminuria" has been used to describe a condition in which, without any symptoms of ill health, variable quantities of proteid matter are present in the urine. The term "functional albuminuria" is a more accurate one to describe this condition. Several varieties of it exist; in some, the proteid matter is only present in the urine on first rising in the morning; in others, only after meals; in others, as a result of severe muscular exertion; and in a considerable number, after cold bathing, and more especially after sea bathing. This functional albuminuria is more especially seen in young adults. The quantity of proteid matter, even in those cases where its presence is detected in the morning or after exertion, is also considerably affected by meals. In the majority of cases of functional albuminuria the amount of proteid matter is small, and requires for its detection the more delicate tests, such as picric acid; and it has been said that approximately 30 per cent. of apparently healthy people may present traces of albumin in the urine as a result of fatigue, diet, or exposure to cold bathing. It is only in a small proportion of cases that the amount of proteid matter is sufficient to give a definite reaction with such a test as cold nitric acid or the heat test; and in a few cases of so-called functional albuminuria the proteid matter present may amount to 1 per cent. Functional albuminuria is sometimes intermittent in its incidence, the urine remaining free from proteid for a variable time, and to some of these cases the term of "cyclical albuminuria" has been given. In most cases of functional albuminuria the urine is free from casts, but it is probable that with more refined methods of examination, such as centrifuging the urine, hyaline casts may be detected in some, and that their presence is not to be taken as absolute evidence of the existence of organic disease. Functional albuminuria has not only to be differentiated from the albuminuria of organic renal disease, but also from what may be termed accidental albuminuria, where the presence of proteid matter in the urine is dependent on the contamination of this fluid with some physiological or pathological exudation. Thus, the urine may contain albumin, from the presence of the discharge of a chronic gleet, or even from the presence of seminal fluid in the urine; and in all cases of supposed functional albuminuria in young adults, the urine should be centrifugalised to determine whether spermatozoa are present or not. Functional albuminuria is probably dependent either on disturbance of the circulation in the kidney, or else it is possible, more especially in the dietetic cases, that the condition is really dependent on the excretion by the kidney of some toxic substance formed in the intestine, perhaps as a result of anomalous digestive or absorptive processes.

Albuminuria dependent on the presence of organic disease must necessarily be due to a lesion of some part of the urinary tract, but the term is more often limited to lesions of the kidney. Where the albuminuria is dependent on disease of the ureter, or of the lower urinary tract, it is simply due to the exudation of inflammatory products or of blood into the urine. When the albuminuria is of renal origin, the lesion determining it is probably primarily in the glomeruli, and it is customary to recognise various causes of the albuminuria, although the essential lesion of the glomeruli is common to them all. Albuminuria of renal origin may be dependent on (1) active or passive congestion of the renal vessels, (2) toxic causes, (3) the organic lesions associated with gross renal disease. In all these cases, however, the real cause of the albuminuria is the damage to the epithelium of the glomerular tuft. The quantity of proteid matter present in the urine may vary from a trace to as much as 40 grms. per dicm. In toxic and febrile albuminuria, and in the albuminuria of renal cirrhosis, the quantity lost is usually small. In active and passive congestion the percentage amount of albumin is often high, amounting to 5 per cent. or even 6 per cent.; but the total quantity lost is usually not very great, owing to the great diminution in the quantity of urine. In chronic Bright's disease and in amyloid degeneration, larger quantities of albumin are usually lost, and it is in some forms of chronic Bright's disease that the daily excretion of albumin may amount to as much as 40 grms., since in this disease the percentage may be high and the quantity of urine secreted considerable. In renal diseases the proteids present in the urine are usually a mixture of serum albumin and serum globulin. Traces of albumose are, however, not uncommonly present, and in rare instances, more especially in renal cirrhosis, considerable quantities of albumoses are excreted. Albumoses are also found, in traces, in diseases associated with the presence of suppuration, such as empyema; or in maladies accompanied by copious inflammatory exudation, as in pneumonia. In these cases the albumosuria is dependent on the formation of albumoses by the activity of the micro-organism causing the initial disease, and the absorption of these materials in the blood, and their subsequent excretion by the kidney. Albumoses in very large quantity are occasionally found in the urine, associated with bone disease, more especially osteomalacia and malignant disease of the medulla of the bones, so-called myeloma. It is possible that the albumosuria of renal disease, such as renal cirrhosis, is dependent on the absorption of albumoses from the alimentary canal and their subsequent excretion. In some of the cases of myeloma and so-called myelopathic albumosuria, renal cirrhosis has been found associated with the bone lesion. In pyelitis the urine will necessarily contain albumin from mere admixture with pus, and in some instances it may be difficult to determine whether the albuminuria is entirely dependent on the pyuria, or whether in addition there is some renal complication. In all such cases, when the urine is allowed to settle so that the pus is deposited, if the supernatant urine contains a large quantity of albumin, there is great probability of the existence of some renal complications, such as Bright's disease or waxy kidney. Where the albuminuria is of renal origin, the urine will, in addition, contain casts, hyaline, granular, and blood casts, and frequently casts may be found containing definite renal elements.

The heat test is that which is most commonly performed for the detection of albumin in the urine; and, in order to avoid fallacies, it is essential that the reaction of the urine should be tested prior to heating, and if alkaline or neutral, that it should be acidified with acetic acid. It is necessary to avoid over-acidity of the urine, and therefore it is advisable not to use a strong mineral acid for acidification. The principal fallacies of the heat test arise either from over-acidification and the non-deposition of small quantities of proteid matter under these circumstances, or else from the urine not being sufficiently acid, and the phosphates undergoing precipitation on heating. The phosphatic precipitate can be distinguished from that caused by albumin, by the fact that the albumin is particulate, and the phosphatic cloud disappears on acidification.

Cold nitric acid test.—The nitric acid must be pure, and should be placed in the bottom of the test tube, and the urine floated on. If proteid matter is present in quantity, a ring is formed at once, but if a trace only is present, the ring only appears on standing. This test is a very accurate one, but is not quite so delicate as the heat test. In concentrated urines, a crystalline precipitate of nitrate of urea may be formed at the junction, but the appearance of this is so different from that of a proteid cloud, that error is scarcely possible. The urine sometimes contains the chromogens of skatol and indol pigments, and this nitric acid test will in such cases cause the appearance of a coloured ring, which has been confused with the presence of proteid. The nitric acid test is especially of value, from the fact that albumoses can be recognised by its aid, the albumoses not being precipitated on heating, and so escaping detection by the heat test. Hetero-albumose is especially characterised by being precipitated by nitric acid, and the precipitate so formed dissolving on heating the fluid. If the presence of albumoses is suspected in the urine, it is better to test the fluid by dropping nitric acid into it, until a well-marked precipitate is formed, which will then disappear on heating and reappear on cooling. If traces of albumose are present mixed with the other coagulable proteids, it will be necessary to remove the latter by boiling before testing the fluid for albumoses. Resinous bodies, besides such as copaiba and oil of turpentine, are excreted by the urine, and the addition of nitric acid to such a urine may cause a precipitate.

Picric acid.—A cold saturated solution of picric acid will cause a precipitate of proteids from the urine. It is a very delicate test, much more so than either the heat or the nitric acid test, but it will cause a cloud when alkaloids are present in the urine.

Salicyl-sulphonic acid.—A cold saturated solution of salicyl-sulphonic acid in water is employed and added gradually to the suspected urine, drop by drop, and the fluid shaken. If any considerable quantity of proteid matter is present, an immediate precipitate occurs. The test is intermediate in delicacy between the nitric acid test and the heat test. This reagent will also serve to detect albumoses, since they yield the same reaction with this reagent as they do with nitric acid. Salicyl-sulphonic acid does not cause any precipitate when alkaloids are present in the urine, nor when resinous matters are present.

Copper sulphate and potash.—Copper sulphate and potash can be employed to detect the presence of albumoses in the urine, as they yield a rose-pink colour, and the ordinary proteids a blue colour. To detect traces of albumose in the urine, it is necessary to remove the ordinary proteids, either by heat or by precipitation. The neutral salts may be used for precipitating, and for separating the various proteids which may be present in the urine. Saturation of the urine with ammonium sulphate will precipitate all proteid matter, with the exception of true peptone. Magnesium sulphate may be used to precipitate serum globulin alone. At the present time these methods are mainly employed for purposes of research, and are of no great use clinically.

To estimate the amount of proteid matter present, the most accurate method is to add 5 c.c. of urine to 50 c.c. of boiling absolute alcohol, and to place the mixture in a hot air oven at 80 ° C. for two hours. The precipitate can then be collected on a weighed filter and washed with alcohol, ether, and water, and weighed after drying in a hot oven at 120° C. A rough estimation of the quantity of proteid matter present may be made by boiling the acidified urine in a test tube, and estimating the depth of the deposit of the proteid matter that occurs. Esbach's method, where the urine is precipitated with a solution containing picric acid, and the amount of proteid estimated by the depth of the deposit in a graduated tube, may also be employed; but neither the Esbach method nor the heat subsidence method gives results which are at all accurate.

Casts.—In all cases of albuminuria the urine should be examined for casts, and if the quantity of albumin is at all small it is advisable to centrifugalise the urine before examining it microscopically for the presence of casts. The significance to be attached to the presence of casts depends a great deal on their nature. Hyaline casts are present in most forms of albuminuria, and may be due to such a trivial cause as the irritation produced by the presence of uric acid. Granular casts are more often associated with the presence of nephritis and Bright's disease, and epithelial casts containing definite renal elements afford clear evidence of the presence of a structural lesion. Blood casts may be dependent on any condition giving rise to hæmorrhage from the kidney substance, and are not necessarily evidence of the existence of grave organic disease. Fibrin casts are sometimes found under similar circumstances, and pus casts are rare, but may be seen in conditions of renal suppuration, such as pyelonephritis and renal abscess. Fatty casts are also sometimes present, and afford evidence of grave degeneration of the renal structures. Waxy casts have been described in amyloid disease, but in many cases the casts described as waxy are really colloidal, and may be found in a considerable number of infective maladies, as in tuberculosis, syphilis, carcinoma. It is probable that flat ribbon-shaped casts are sometimes formed in the ducts of mucous glands situated in the mucous membrane of the genito-urinary tract, and such casts are in no way significant of the existence of renal disease.

Pyuria.—Pus may be present in the urine, either as a result of disease of some portion of the genito-urinary tract, or else as a sequel to the rupture of an abscess into this tract. Care must always be taken that the presence of pus in the urine is not merely due to admixture from inflammation of the urethra or the vagina. Before investigation, the urine should be passed in two portions, as in this way it is often possible to determine that the pus is derived from the urethra if it is present in the first portion. In all cases of pyuria the pus should be separated from the urine by deposition or by centrifuging, and the supernatant urine should be tested for albumin, since, if gross renal disease be present, this fluid will probably contain a larger proportion of albumin than can be accounted for by simple admixture with pus. Urine containing pus may be acid or alkaline. The former is more characteristic of pus of renal origin, and the latter if the pus be derived from the bladder; but in some cases of cystitis the urine may still be faintly acid, and in cases of pyelitis, especially where it is secondary to cystitis, the urine may be alkaline. In many cases of abscess of the kidney, especially where this is of embolic origin, the pyuria may be sudden and intermittent. Pus is not necessarily present in all suppurative diseases of the kidney, and this is more especially true of pyonephrosis, where the obstruction to the ureter may be such as to prevent any of the pus reaching the bladder. The presence of pus in the urine is best recognised by microscopic examination, but the addition of liquor potassæ to the suspected urine will cause it to become ropy. The addition of ozonic ether to the suspected urine will cause an effervescence.

Chyluria.—The urine in exceptional instances is milky, from the presence of chyle, and in some cases this may be so highly albuminous as to lead to the formation of a soft trembling milky clot, either before or after its passage. In chyluria the fatty matter in the urine is in a state of extreme subdivision, the globules of fat being very much smaller than those present in milk. Shaking the urine with ether will commonly cause the milky appearance to disappear, but frequently repeated shaking with fresh portions of ether are required, owing to the extreme state of subdivision in which the fat is present. Chyluria is apparently dependent on some communication existing between the lymphatics and the urinary channels, as for instance in the bladder, and perhaps in the ureter, and it is most often associated with obstruction of the lymphatic system, caused by the presence of filaria, acquired during residence in a tropical climate. Some cases of chyluria, however, are occasionally seen in individuals who have not lived abroad, and where examination of the blood fails to reveal the presence of filaria. The chyluria is not uncommonly intermittent in its presence, being sometimes only found in the urine secreted during the night, and in exceptional instances it is only seen as a result of the assumption of some particular posture. Thus cases are on record where chyluria has only been present when the patient lies on one side, and not on the other. As far as is known, chyluria is always dependent on obstruction to the lymphatics, and is probably due to the rupture and leaking of the distended lymphatics into some portion of the urinary tract. The presence of chyle in the urine is determined by its milky appearance, its microscopical appearance, and the solution of the fatty matter after prolonged shaking in ether.

Bacilluria.—In microbic diseases, organisms causing the disease are frequently excreted in the urine, in addition to the toxines elaborated by the activity of these organisms. In typhoid fever the urine not uncommonly contains the typhoid bacillus, and most observers are agreed that the typhoid bacillus is more readily recognised in the urine than in the fæces.

The *Bacillus coli communis* is also not uncommonly present in the urine; in tuberculous disease of the kidney or of the urinary tract the bacillus of tuberculosis is found, and it has been asserted that this organism may be present in the urine as a result of tuberculous disease in other parts of the body without the presence of tuberculous kidney. The smegma bacillus, however, presents great resemblances, both in its appearance and in its staining reactions, to the bacillus of tubercle; and it is often necessary, in order to determine with certainty that the tubercle bacillus is present in the urine, to inoculate guinea-pigs with the suspected material, and not to rely simply on the staining reactions of the organism.

MALFORMATIONS AND DISPLACEMENTS.

Anomalies of the kidney are of importance in medicine, partly owing to the errors of diagnosis which are liable to occur as the result of the non-recognition of a displaced or malformed kidney, and partly owing to the fact that abnormal kidneys are more liable to undergo certain pathological changes. This is seen in the frequency with which displaced or malformed kidneys may become affected with inflammatory diseases. Anomalies of the ducts also may exert an influence in the production of hydronephrosis, and may be of importance as predisposing causes in such conditions as pyelitis. Anomalies of the kidneys may be divided into those in which the organ is displaced, those in which the organ is malformed, and those in which the number of kidneys is abnormal. Anomalies in shape are, however, closely associated with anomalies in number

ANOMALIES IN NUMBER.

Both kidneys may be absent; inasmuch as this condition is incompatible with life, it is only of embryological interest. In very rare cases a supernumerary kidney may be present, but this condition is not so common as the presence of supernumerary suprarenals or supernumerary spleens.

The usual anomaly in number is for only one kidney to be present, and, as mentioned above, this anomaly is very often associated with some malformation in the shape. Where only a single kidney is present, this condition may be due to one of the three following causes:—One kidney may be congenitally absent; or, secondly, two kidneys may have undergone fusion; or, thirdly, two kidneys may have been present in early fœtal life, but, as the result of pathological changes occurring during fœtal life, one may have undergone atrophy. Further, the presence of a single kidney in adult life may be dependent on atrophy of its fellow having occurred during early life. In cases of single kidney, dependent on want of development of its fellow, no traces of the renal artery or ureter can be found on the side where the kidney is absent. In conditions where the kidney has undergone extreme atrophy during fœtal or post-fœtal life, some small remnant of the kidney and of the renal vessels and ureter may be found, and, where such an atrophied kidney is present, it may be found that the kidney has been developed in an anomalous manner, such as, for instance, that it may contain no glomeruli and no convoluted tubules. When a single kidney is present, either as the result of the absence of its fellow or as a sequel to its early atrophy, the opposite hypertrophied organ is frequently more than twice the weight of the normal kidney. Such hypertrophied kidneys may weigh as much as a pound, or more. In most cases the presence of a single hypertrophied kidney in no way prejudices the life of the individual, and many such cases have lived to extreme old age. On the other hand, Bright's disease and calculous disease are not very uncommonly associated with the presence of a single kidney.

Atrophy of one kidney occurring in fœtal life would seem to be sometimes dependent on an anomaly in the development of the organ, and in such cases the glomeruli and convoluted tubules may be absent; in other cases, occurring in fœtal or in post-fœtal life, it is certainly dependent on disease involving the renal artery, leading to its occlusion; and in others it is associated with, and dependent on, obstruction to the ureter, due to the presence of calculi or some other morbid process.

The presence of a single kidney is often dependent on the fusion of two primarily separate organs; most frequently this fusion occurs in such a manner that the two lower extremities of the kidney are joined together across the front of the vertebral column by a mass of renal tissue forming the well-known horse-shoe kidney. Such kidneys are generally malformed in other respects, so as to be long and narrow, and the lobulation is extraordinarily well marked.[1] The band of renal tissue joining the two kidneys usually crosses the spinal column at the level of the second and third lumbar vertebræ, and joins the lower extremities of the two kidneys. The horse-shoe kidney has usually an anomalous blood supply, and the two ureters as a rule pass over the front of the mass on their way to the pelvis. In some cases the appearance of the horse-shoe kidney is spurious, the band of union between the two kidneys consisting only of fibrous tissue. The horse-shoe kidney is the commonest form of the fused kidney. The other varieties of it—(*a*) where the two kidneys are united end to end, and (*b*) where they are fused by their surfaces becoming adherent, are much rarer; the former has been named the sigmoid kidney, and the latter the disc-shaped kidney (Newman). In both these conditions the ureters are found on one side of the body, but one subsequently crosses over to the other side, so that the two ureters open in their normal situations in the urinary bladder. The horse-shoe kidney is of some clinical importance, owing to the fact that the mass of renal tissue passing over the front of the vertebral column may be mistaken for a tumour. The other two forms are only of pathological and embryological interest. In very rare cases the horse-shoe kidney has caused thrombosis of the vena cava by its pressure on this vessel, and the arrest of the circulation has caused death.

ANOMALIES IN POSITION.

The kidney may be displaced, either congenitally or as the result of disease. Congenital displacements are divisible into those in which the kidney is fixed in its anomalous situation, and, secondly, those where there is displacement with mobility. The most important fixed displacements are those in which the kidney occupies a lower level than that normal to it, such as, for instance, over the sacro-iliac synchondrosis, in the concavity of the sacrum, or in the iliac fossa. Displacement over the sacro-iliac synchondrosis is the most common. Such anomalies in position usually affect only one kidney, and they are not uncommonly associated with anomalies in the development of the large intestine, and in some cases they are associated with imperforate anus. They are of no great clinical importance, but it has occasionally happened that a displaced kidney has undergone enlargement, owing to kinking of the renal vein or ureter, and the swollen kidney has given rise to severe symptoms, together with local pain and tenderness. If the swollen kidney is in a situation accessible to clinical examination, *e.g.* the iliac fossa, it may be mistaken for such an inflammatory condition as appendicitis. Where the organ is displaced into the concavity of the sacrum, it may be mistaken for a tumour.

Displacements with mobility are far more common, and give rise to the condition which is known as movable kidney.

MOVABLE KIDNEY (NEPHROPTOSIS).

Some authorities recognise two varieties of movable kidney, one where the organ is only slightly movable, and to this condition some restrict the term of movable kidney; the other where the mobility is greater, and to this the term floating kidney has been applied; it is, however, difficult to draw a hard and fast line between the two conditions. Normally the kidneys are only slightly movable on respiration, and this, in the case of patients with a lax and thin abdomen, can be readily detected on palpation. The mobility of the normal kidney during respiration is not sufficient for the hand to be pushed above the kidney, and some would consider the condition pathological if more than approximately the lower third of the kidney can be felt during deep respiration. It is, however, extremely difficult from the mere extent of movement to separate the truly movable kidney from degrees of movement of the organ compatible with health. The anatomical distinction between movable kidney and floating kidney lies in the fact that in the latter case the peritoneum passes behind the kidney, to a varying extent, forming a mesonephros. In simple movable kidney the organ slips about behind the peritoneum. Even where a mesonephros is present, it does not necessarily follow that the condition is congenital, as it is probable that a spurious mesonephros may be formed, as a result of acquired mobility of the organ. The normal kidney is kept relatively fixed, partly by the pressure of the surrounding viscera, largely by the tonus of the abdominal muscles, and also, to a great extent, by the fatty bed in which it lies, inasmuch as this is traversed by bands of fibrous tissue, which some anatomists have described as ligaments. In addition, the reflexions of the peritoneum from the anterior surface of the organ to the surrounding viscera serve to fix it. The peritoneum passing from the upper part of the anterior surface of the kidney to the duodenum in the vicinity of the bile duct, is of some importance in this relation.

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[1] Lobulation to a varying extent is seen in all kidneys, but in some cases the fœtal condition of extreme lobulation persists to a marked extent.

Etiology.—Movable kidney is more common in women than in men, in the proportion of seven to one. Some authorities have considered it so prevalent in women as to amount almost to a normal condition in its slighter forms. This, however, arises from the extreme difficulty of distinguishing between the normal mobility and the slighter forms of abnormal mobility. Probably the distinction should be drawn quite apart from the presence of symptoms, in the fact that in the pathological condition the downward displacement is such that the hand can get above the kidney. If this definition is allowed, the condition is by no means so common. Different authorities state that from 3 to 25 per cent. of women suffer from movable kidney. Movable kidney is more common on the right side than on the left. The most important predisposing causes are emaciation and laxity of the abdominal walls, arising sometimes simply as the result of general malnutrition; in others, as a sequel to distension of the abdomen, produced by pregnancy or other causes. Laxity of the abdominal walls may predispose, not only to preternatural mobility of the kidney, but also to mobility of other organs, such as the liver and intestines (Glénard's disease). Tight lacing is also unquestionably a predisposing if not an exciting cause of the condition. The most important exciting cause is traumatism. The kidney may become displaced and preternaturally mobile, as the result of dislocation of the organ following jumping. Direct injury to the loin may also be a cause.

Morbid anatomy and pathology.—In the slighter forms of the condition the kidney is simply preternaturally loose, and will slip about behind the peritoneum. In the more severe forms, with the presence or development of a partial mesonephros, the mobility of the organs is such that it may be more or less freely moved about in the abdominal cavity

The displacement and mobility of the kidney may produce other changes in adjacent viscera, and more especially the stomach. Dilatation of the stomach, apparently dependent on the traction exerted on the duodenum and pylorus, through the peritoneal reflections from the kidney to the duodenum, is not uncommon. The bile duct may also be dragged on or kinked, and the renal vein and ureter are also specially liable to be partially obstructed or kinked as a result of the mobility of the organ. The right kidney is more usually affected than the left, and both are affected in a small minority of cases.

Symptoms.—In the great majority of cases of movable kidney no symptoms are produced, and the condition is discovered accidentally; in some, however, a great variety of symptoms are developed, and in a few the effects may be serious. In a large number of instances a condition of neurasthenia is present, but whether this is produced by the mobility of the kidney, or whether the two conditions are simply associated, is a matter on which there is great difference of opinion. In many such cases the neurasthenic symptoms are sufficiently serious to produce not only a nervous, excited, and tremulous condition, but sleep may be interfered with, and such patients often become emaciated. A very constant symptom of movable kidney is pain; the pain is usually in the abdomen and in the loin, and only rarely has either the course or the severity of that seen in renal colic. In some cases, however, the pain is much more severe, and may simulate that produced by renal colic, and, like it, may be accompanied by intense nausea and vomiting, and there may be also tenderness of the loin and abdomen over the affected kidney. In some patients the attacks of pain are paroxysmal, and when these are accompanied by nausea and vomiting, they have been known as Dietl's crises. In others the pain is accompanied by considerable abdominal tenderness, and some abdominal distension, so as to simulate intestinal obstruction. In the great majority of cases of movable kidney, no other symptoms are produced; but in some hydronephrosis, persistent or intermittent, may be produced, owing to kinking of the ureter. In others severe pain, accompanied by hæmaturia, may occur, and it is supposed that in such cases the renal vein becomes kinked, causing intense congestion of the organ. The development of intermittent or constant hydronephrosis is the most important of the more serious results of movable kidney, and it is often associated with some slight anomaly of the ureter—such as, that it is preternaturally narrow, or that it enters the renal pelvis at an abnormal angle. The interference with the ureter causing hydronephrosis, may result in rare instances from the presence of anomalous renal arteries. In some cases of movable kidney gastric symptoms are present, such as nausea, distension, palpitation after meals, and vomiting; in fact, many of the symptoms which are associated with dilatation of the stomach—and it is unquestionable that dilatation of the stomach is sometimes associated with movable kidney, and may, as mentioned above, be dependent on the traction exerted on the duodenum and stomach, through the peritoneal reflexions passing from the kidneys to these organs. Jaundice has also been described, and may be dependent on traction on the bile duct producing obstruction.

Diagnosis.—The diagnosis of movable kidney is determined by the detection of the anomalous mobility of the organ by palpation with one hand in the loin and the other over the anterior surface of the abdomen. In many cases it is possible to feel the kidney slip down on deep inspiration, simulating a piece of wet soap; and in cases of excessive mobility it is by no means difficult to grasp the organ, and to keep it down. Palpation of the organ usually causes pain, and may produce sensations of nausea and faintness. There is not as a rule much difficulty in the detection of the condition, but it is not uncommon to overrate its importance, and in cases of general neurasthenia to attribute to the mobility of the kidney far greater importance as an etiological factor than it deserves. Errors of diagnosis may arise in cases of chronic indigestion, if movable kidney is not thought of as a possible cause. In cases where serious effects, such as hydronephrosis, are produced, there is usually no difficulty in the diagnosis. In some cases the condition may be confounded with renal colic and calculous disease, owing to the similarity of the pain and the presence of hæmaturia.

Prognosis.—The condition in the majority of cases is of no great importance. In a few, however, and more especially where hydronephrosis is produced, or where the attacks of pain are very frequent, the general health of the patient may suffer seriously.

Treatment.—In the slighter forms of movable kidney probably no treatment is required. In most cases the application of a suitable pad, and treatment directed to improve the general health, more especially by increasing the fat and the nutrition of the body generally, and improving the condition of the abdominal muscles, are of great importance. In some such cases Weir Mitchell treatment may be of service, but massage must be applied with great discretion, as it is by no means impossible for the condition to be aggravated by massage. If the symptoms are very severe, and are not relieved by a belt and pad, surgical advice must be sought, and in some cases nephrorrhaphy may be of service. In the most severe cases, where constant or intermittent hydronephrosis and very severe pain is experienced, it may be necessary to incise or to remove the affected organ; this, however, is very rarely required.

ANOMALIES OF THE URETERS.

Anomalies of the ureters, although frequent, are of no great pathological importance. In some cases, however, owing to extreme narrowness of the ureter, or owing to its mode of attachment to the pelvis of the kidney being anomalous, it may be a cause of hydronephrosis. In rare cases the ureters are found congenitally greatly dilated, and this anomaly of the ureter is usually associated with malformation of the kidney, with excessive development and dilatation of the pelvis of the kidney; and some cases of this kind have died as the result of an ascending inflammation of the kidney.[1] The other anomalies of the ureters, such as double ureters, are of no clinical importance.

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[1] The writer has seen three such cases, where there has been a history of incontinence of urine from childhood, and where ultimately uræmic symptoms, like those seen in pyelonephritis have supervened, and caused death. In no case could any cause be detected for the dilatation of the ureters, but in all the kidneys were malformed, in addition to presenting the usual acute lesions seen in pyelonephritis.

STRUCTURAL DISEASES OF THE KIDNEY.

CONGESTION.

Congestion of the kidneys may be active or passive. Active congestion is a condition difficult to separate from nephritis, although it has been supposed that acute and sudden distension of the blood vessels of the kidney may occur under a number of conditions, apart from the subsequent development of nephritis. Usually, however, active congestion is merely a stage in the process of nephritis.

Passive Congestion.

Etiology.—Passive congestion may be seen in any condition in which the return of the blood through the venous system is impeded. In most cases the obstruction causing the venous engorgement is in the heart or lungs, sometimes in the inferior cava, and more rarely in the renal veins themselves, although it is in the last condition that the most extreme examples of passive congestion are found. Diseases of the lungs, such as emphysema and bronchitis, causing hypertrophy and dilatation of the right side of the heart, are especially liable to cause passive congestion of the kidneys. Mitral disease, and especially mitral regurgitation, is a very common cause of passive congestion of the kidney; and the changes produced in the kidney in this disease have sometimes given rise to the name of "the cardiac kidney" being applied to this condition. Mitral disease cannot, however, produce renal congestion until its effects have been sufficiently severe to cause tricuspid incompetence. Adherent pericardium and thickening of the pericardium may also impede the return of the blood, by causing stenosis of the inferior vena cava at its entry into the pericardium.

In cirrhosis of the liver, passive congestion of the kidney is not uncommon, and may be dependent on partial stricture of the vena cava, where it lies in the notch in the posterior border of the liver. It has been thought by some that the mere accumulation of ascitic fluid in the abdominal cavity might be a sufficient cause of the renal congestion often seen in this disease. It is probable, however, that in many cases the obstruction to the renal circulation is really brought about by obstruction to the cava, produced where the vein lies in the notch on the posterior border of the liver, and in some cases the caval obstruction so produced is extreme. Other abdominal diseases, such as enlarged glands, tumours, hydatid cysts, and possibly pregnancy, may also press on the renal veins, and cause renal congestion. Thrombosis of the renal vein may occur as a result of a general marasmic condition, and is sometimes seen, not only in marasmus, but also during the asthenia following acute disease, *e.g.* diphtheria. Thrombosis of the renal vein may also complicate other morbid processes in the kidney, such as interstitial nephritis and surgical kidney; and it is also occasionally seen in the granular kidney, and is not uncommon in malignant disease of the kidney. Malignant growths in other regions of the abdomen may, however, spread into the lumen of the cava, and in that way obstruct the renal vein. Partial thrombosis of the renal vein, associated with phlebitis, may occur as a sequel to suppuration in the pelvis, a clot being formed in one small vein, and gradually extending up the vena cava until it may involve the renal veins. In rare cases, a spreading thrombosis may be found involving the pelvic veins, the vena cava, and the renal veins, as a sequel of abdominal operations, where a vein has been tied in a pedicle, *e.g.* in oöphorectomy, and where there may be no obvious post-mortem signs of the existence of septic mischief, such as the presence of pus.

Morbid anatomy.—In passive congestion of the kidney, the organ is enlarged and red, and the vessels obviously distended. The congestion is especially marked in the pyramidal portion, and streaks of congestion may be seen passing upwards through the pyramids. The Malpighian bodies undergo a certain amount of distension, but not so great as that seen in active congestion. Where the obstruction has existed for some time, the kidneys are tough and hard; and on stripping the capsule a slightly granular surface may be left; exceptionally where the fibrosis is excessive, the organ may be somewhat smaller than normal. On microscopic examination in recent cases, the veins and capillaries are found gorged with blood; and extravasations of various size have occurred in the interstitial tissue between the tubules, especially in the medullary portion of the organ. In cases of extreme congestion, the tubules themselves may be filled with blood. In cases of long-continued, slight congestion, as in cardiac disease, there may be a considerable increase in the connective tissue, both between the tubules and round Bowman's capsule. The walls of the tubules, or rather the basement membrane, is also thickened, and much more obvious than normal. The epithelium of the tubules in places has undergone changes, so that in both the convoluted and the straight tubules it may have lost its typical character, and the tubules may be lined with clear cubical cells. In many cases of passive congestion of the kidney, however, where more extreme tubular changes are found, they are in part dependent on the supervention of inflammatory complications; and in some instances it may be difficult to distinguish between the changes in the epithelial structures due to passive congestion and those seen in the chronic forms of Bright's disease.

Symptoms.—The most characteristic symptom of passive congestion of the kidneys is a diminution in the quantity of urine passed, often less than 20 oz. per diem. The urine is dark in colour, the specific gravity is high, generally as much as 1025 or more, and copious deposits of lithates are usual. Albumin is present in varying amount, sometimes not more than a trace, more usually in such quantities as one-sixth or one-eighth, occasionally as much as one-third. It is probable that in simple passive congestion of the kidney, larger quantities than these are not seen, and that the highly albuminous urine, which is sometimes associated with pregnancy (where the urine may contain half or even more albumin), is not dependent on simple passive congestion. The albuminuria of pregnancy is more probably of toxic origin. Blood is often present in variable amount in passive congestion, and blood casts and hyaline and granular casts are common. Dropsy is frequently present in passive congestion of the kidney, but it is of the cardiac type, being most marked in the lower extremities.

Diagnosis.—The main difficulty in the diagnosis of passive congestion is to separate this condition from active congestion and from Bright's disease. It is often extremely difficult in cases of mitral disease to determine whether the patient is suffering from mitral disease, complicated by passive congestion of the kidney, or whether Bright's disease is present. Blood, albumin, granular and hyaline casts are found in both conditions, although in Bright's disease the presence of casts containing definite renal elements will often afford a means of recognising the more serious renal lesion. Usually in Bright's disease the urine is pale, and in passive congestion dark; but in Bright's disease, complicated with cardiac dropsy due to a failing heart, the specific gravity of the urine may be high. The most important points as regards the urine, in effecting a differential diagnosis, are the smaller amount of albumin and the higher specific gravity, and the presence of only granular and hyaline casts in passive congestion. Useful confirmatory evidence may often be obtained by observations of the state of the pulse and of the condition of the retina.

Prognosis.—The prognosis of passive congestion of the kidney depends chiefly on its cause. In cardiac disease the outlook is far better than if actual disease of the kidney, such as Bright's disease, is present. Passive congestion of the kidney, however, materially increases the gravity of such diseases as emphysema, cardiac disease, and hepatic cirrhosis.

Treatment.—The treatment of passive congestion is really the treatment of the cause of the condition; in cardiac cases, and especially in mitral disease, it is very amenable to such agents as digitalis, which act as diuretics, by improving the efficiency of the general circulation. Caffeine may also be of service, although the action of this substance is one that commonly produces only temporary relief, the diuretic effect being far better marked with initial than with subsequent doses. Caffeine has not the persistent beneficial action that digitalis has. In addition to improving the circulation by such drugs as digitalis, strophanthus, etc., purgation, especially with saline purgatives, causing copious watery stools, is of service. The warm bath, hot air bath, and simple diuretic drinks are of service. It is doubtful whether dry cupping the loins is of much value.

The diet must be a simple one, and milk is often valuable from its diuretic action. Stimulants, which are often required for the treatment of the cardiac disease, should be given in small quantities, and diluents should be ordered freely.

ACTIVE CONGESTION.

It is difficult to draw a distinction between active congestion and nephritis, inasmuch as active congestion is the first stage in inflammatory processes involving the kidney.

Etiology.—Active congestion of the renal vessels may be produced—(1) By the excretion of toxic substances ingested, as in the case of cantharides, turpentine, copaiba, cubebs, carbolic acid, quinine, and mineral acids in certain doses. (2) By the excretion of toxic substances, formed in the body as the result of morbid and other processes. It is probable that the albuminuria which frequently accompanies pregnancy is dependent, not so much on the mechanical congestion of the kidney, produced by the uterine tumour, as upon the circulation in the maternal vessels of some toxic substance, the product of disordered metabolism, the excretion of which by the kidney produces albuminuria. In morbid processes, involving distant parts of the body, the toxic substances excreted by the kidney are usually the toxines produced by the organisms causing the disease. In some cases, however, vascular lesions in the kidney may be produced by the excretion of the microbes themselves. (3) There is little doubt that extreme congestion of the kidney may be brought about by an influence exerted on the blood vessels of the kidney through the nervous system. Active dilatation of the renal vessels may be brought about experimentally by excitation of the central end of the lower dorsal posterior roots: excitation of most other afferent nerves tends to cause constriction of the renal vessels. In the human subject it is known that extreme congestion of the kidney may sometimes be the sequel of irritation of the lower urinary tract; and this is perhaps the explanation of the extreme congestion which may sometimes supervene as the result of passing a catheter.

It is supposed, mainly on clinical grounds, that exposure to cold may cause active congestion of the kidney, the action of the cold on the skin producing reflexly extreme dilatation of the renal vessels, and that this acute congestion may not only pass on into nephritis, but may even go further and produce Bright's disease. There is, however, no experimental evidence showing that stimuli acting on the skin are capable of producing renal congestion.

Active congestion and nephritis in the human subject are generally of toxic origin. The toxæmia produced by the acute specific fevers is the most frequent cause of active congestion of the kidney and of nephritis, and the renal lesion occurs generally during the height of the febrile disease; although this complication may occur in any acute specific disease, it is most characteristic of scarlet fever, pneumonia, typhoid fever, and diphtheria, but especially of the first.[1]

Morbid anatomy.—The kidneys are swollen and gorged with blood, and in the more acute forms of the disease they may be increased to twice their size, and it is said that occasionally the distension of the organ may be such as to cause splitting of the capsule. On section, the kidney is not only uniformly red and gorged with blood, but commonly minute points of increased vascularity or even of actual hæmorrhages may be seen. In many such cases the bright red points are merely the excessively distended glomeruli; in others, however, the appearance is dependent on hæmorrhage having occurred into the tubules, and the red dots and streaks are dependent on the presence of the extravasated blood in the renal tubules. The interstitial tissue between the tubules is also infiltrated, the blood vessels distended, and here also minute hæmorrhages may be seen. The signs of vascular enlargement are especially marked in the cortex of the kidney.

The vessels of the glomerular tuft are distended, and Bowman's chamber often contains, not only an albuminous exudate, but also blood corpuscles. In severe cases the glomerular changes are more marked, and the capillary tuft and Bowman's chamber both show signs of proliferation. In the severe forms, where the circulation in the glomerular tuft is more or less arrested, the cells of the convoluted tubules show necrotic changes and may in places be shed. In the slighter forms of the affection, such as are usually seen in febrile diseases, the epithelium of the tubules shows the appearances of cloudy swelling, and the section of the kidney presents to the eye an opaque appearance.

All gradations are found, between cases where cloudy swelling is the main feature, together with some vascular engorgement, and other cases where the inflammatory process is as marked as in acute Bright's disease. In many cases, especially when of septic origin, the changes are more marked in the interstitial tissue—congestion, hæmorrhage, and exudation being all present. In some forms, and more especially when a result of typhoid fever, the exudation in the interstitial tissue is disseminated in its distribution, accumulation of leucocytes occurring in sufficient numbers to produce small masses visible to the naked eye, and simulating growths in appearance. To these cases the term of lymphomatous nephritis has been applied, and the kidneys are usually greatly increased in size, and present to the naked eye a superficial resemblance to the large white kidney, a variety of Bright's disease.

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[1] It is doubtful whether congestion of the kidney may not sometimes be produced by excessive cardiac hypertrophy, *e.g.* double aortic disease.

The lesions in nephritis are not usually equally well marked throughout the kidney, and this is especially the case in the slighter forms. Pathologists recognise several varieties, *e.g.* glomerular nephritis, tubal and interstitial nephritis, according to which element of the kidney is most affected; but this classification is largely artificial, as all the kidney structures are affected—to different extents, it is true. Scarlet fever is especially apt to produce a nephritis, where the most marked lesion is the one affecting the glomeruli. Inasmuch as the blood supply of the convoluted tubules is derived from the glomerular chamber, it is clear that tubal changes must be present in all cases where the glomeruli are seriously damaged.

Symptoms.—In many cases of active renal congestion the patient presents no true symptoms, but only changes in the urine; in others, however, pain in the back may be present, and this is supposed to be dependent on the stretching of the capsule produced by the vascular engorgement. The urine is diminished in quantity to varying extents, and in very severe forms of renal congestion it may even be suppressed; more usually, however, it is simply diminished in amount. It is high-coloured, and frequently contains blood, and in the more acute forms of renal congestion it may contain large quantities of blood. The blood may be present in small quantity, and only in the form of blood casts. The specific gravity is usually high, and the urine contains a high percentage of urea and other solids; owing to the great diminution in the amount of urine secreted, the total amount of urea and other solids excreted is greatly diminished. Albumin is always present, and in the slighter forms of the condition, such as those accompanying febrile diseases like scarlet fever, typhoid, and pneumonia, the presence of albumin in the urine may be the only sign of the renal congestion. The amount of albumin in such cases is not usually great, and in the slighter forms it may only amount to a cloud; more usually the quantity is one-tenth to a sixth. In severe forms of renal congestion, such as are sometimes produced by turpentine or cantharides, there may be larger quantities present.

Hyaline, granular, and blood casts are commonly to be found. In the more serious forms, where the condition passes into that of a severe nephritis, there may also be casts containing definite renal elements, such as renal cells. In the common forms of the disorder, however, such as are seen accompanying the acute specific fevers, the urine is only diminished in quantity, of high colour, and contains small quantities of albumin. In the more severe forms, as mentioned above, where blood is present, the blood is not only present in the form of blood corpuscles, but very commonly also in the form of blood pigment, giving rise to the smoky tint of urine, dependent on the presence of methæmoglobin.

Anasarca is not present in cases of renal congestion, or in cases of nephritis, and this is one of the means by which the condition is diagnosed from acute Bright's disease. It is not only very difficult to separate congestion of the kidney from nephritis, but also to separate some forms of acute nephritis from acute Bright's disease.

Diagnosis.—The diagnosis of active congestion of the kidney or of nephritis is made on the presence of a scanty, high-coloured albuminous urine, arising in the course of one of the febrile diseases or occurring as a result of the action of the toxic causes mentioned above. The absence of dropsy is an important sign in separating such cases from Bright's disease. It is difficult, however, to draw a hard-and-fast line between nephritis and Bright's disease, and some authors regard Bright's disease and nephritis as almost identical terms. It is, however, essential to separate the albuminuria that arises during the height of febrile diseases, and dependent on toxic causes, from the very much more serious condition of Bright's disease, which may occur as a sequel to many of the febrile diseases which during their height cause a transitory nephritis.

Prognosis.—The prognosis of active congestion of the kidney and of nephritis depends very largely on its etiology as well as on its severity. The transitory nephritis seen in the course of scarlet fever, typhoid, pneumonia, and other diseases, is usually of no great importance, and disappears with the subsidence of the fever of the original malady. On the other hand, in some cases it passes on into Bright's disease; and this may be seen, for instance, even in such a disease as diphtheria as a rare complication. More usually, where true Bright's disease supervenes as a result of some previous acute disease, it appears later in the course of the original disease, and more especially during convalescence, after the subsidence of the acute illness, and in such cases the transitory nephritis present during the height of the disease may disappear, although the graver lesion subsequently develops. In the case of the nephritis dependent on such toxic substances as turpentine or cantharides, although the condition may completely subside, and the urine regain its normal character, it is probable that the damaged kidney is more prone to become subsequently affected by Bright's disease than the normal organ is. The albuminuria of pregnancy, which may also, as mentioned above, be dependent really on a toxic cause, frequently clears up with the termination of the pregnancy, but in some cases this condition produces very serious symptoms, and may even cause death with uræmic phenomena.

Treatment.—The treatment of active congestion involves necessarily the treatment of the disease causing the condition, but the renal lesion necessitates care in the selection of the drugs employed in the treatment of the primary disease. Patients suffering from renal congestion should not, under any circumstances, be blistered by means of cantharides, and many authorities consider that such drugs as mercury and opium should not be administered in such cases. The dangers, however, of opium and morphine have been exaggerated in such cases, and the mere presence of albuminuria during the height of a febrile disease, such as typhoid or scarlet fever, should not be held to contra-indicate the use of a drug. Diluents and simple diet should necessarily be ordered, and frequently hot applications to the loin, or even cupping or leeching, may produce relief to the pain sometimes experienced. In other respects, and especially in the more severe forms of nephritis, the treatment should be similar to that ordered in acute Bright's disease.

EMBOLISM.

Infarction of the kidney vessels may result from the detachment of fragments of clot from the interior of the heart, or from aneurysms of the great vessels, and also may result from fragments of calcareous matter detached from atheromatous ulcers of the aorta. One of the most important varieties of renal embolism is that caused by the lodgment of micro-organisms in the capillaries of the kidney.

Etiology.—Infarction of the kidney occurs frequently in cases of heart disease, and most frequently in cases of malignant endocarditis. It is, however, not restricted to this condition, but may result during the course of simple endocarditis, either from the detachment of blood clot from the vegetations on the valves, or else from the detachment of fragments of blood clot from the cavities of the heart, more especially from the left auricle. It is common both in aortic disease and mitral disease. As mentioned above, it is occasionally seen in the course of aneurysms involving the thoracic aorta, and also in cases of atheromatous disease of this vessel.

Multiple infarcts of the kidney, with the formation of abscesses, are a very common accompaniment of pyæmia.

Morbid anatomy.—The arrangement of the vessels in the kidney is such as to render the organ one where the effects of arterial occlusion from infarction are singularly well marked, owing to the arterioles of the kidney being end arteries, and anastomosing but little with their fellows. As a result of this, the occlusion of a renal artery, either large or small, causes the necrosis of a more or less wedge-shaped area of the cortex. In many cases, however, the area of the cortex affected is somewhat irregular, and it may be even quadrilateral. Unless the vessel occluded is a very large one, the medullary portion of the kidney is not involved to any great extent.

The appearances of the infarct and the result depend very largely on the nature of the agent that is occluding the renal vessel. If it consists of a non-irritating material, such as a fragment of blood clot or a portion of calcareous plate, the effects produced are merely mechanical and similar to those seen experimentally as a result of the ligature of a branch of the renal vessel. The portion of the kidney in which the circulation is arrested undergoes necrotic changes, and assumes a deep yellow colour; this is surrounded by a zone of congestion, which is held to be dependent on a back flow through the veins from the adjacent portions of the kidney where the circulation is still intact. Where the occluded material contains micro-organisms, other changes result, and the central portion of the necrotic area may undergo softening with the formation of an abscess. In other cases, more especially where embolism results from the lodgment of microbes, suppuration occurs, without the formation of any very large infarct, and in this way multiple abscesses of the kidney may arise. In cases of simple infarction of the kidney, where no suppuration results, the necrotic area is slowly absorbed, and ultimately a depressed scar remains to mark the site of the former infarction.

In some cases, and especially in heart disease, repeated and extensive embolism of the kidney may occur, and such organs may be greatly deformed through the formation of scars.

In the early stages of an infarct, that is to say, in the first two or three days after its formation, the kidney area affected is of a deep red colour, owing to the back flow of blood from the adjacent areas. In a very few days, however, the necrotic changes occur which cause the tissue to assume the characteristic yellow appearance, and this may persist for many weeks, so that it is difficult to gauge accurately the age of an infarct by its naked-eye appearances.

Symptoms.—Infarction of the kidney frequently occurs without producing any symptoms drawing attention to the nature of the lesion. In other cases the symptoms are marked, and the condition can be diagnosed.

A sudden attack of pain in the loin, accompanied by changes in the urine, and in the infective cases with the occurrence of rigors or of an initial rigor, are the most characteristic symptoms. Where the infarction results from the detachment of a portion of blood clot containing no infective agent, the rigor and the pain may be absent, and the changes in the urine afford the only means of surmising the occurrence of infarction.

The nature of the urinary changes depends largely on the nature of the infarction; if simple, as in the case of detachment of blood clot, a sudden albuminuria or a sudden increase in an existing albuminuria may be the only sign pointing to its occurrence. In other cases, in addition to the occurrence of sudden albuminuria, an equally sudden hæmaturia may result. In the septic cases, a few days after the occurrence of a rigor, and perhaps of pain in the back, the urine may suddenly contain a considerable quantity of pus. Although the sudden occurrence of hæmaturia, associated with pain, is the most characteristic phenomenon produced by renal infarct, in a very large majority of cases the infarction simply causes the occurrence of a sudden albuminuria. If the infarction is not repeated, the albuminuria, hæmaturia, or pyuria caused by its occurrence rapidly subsides, and the urine regains its normal composition.

Diagnosis.—The main interest of renal infarction lies in its diagnosis, inasmuch as it is liable to be confounded with nephritis, and with passive congestion of the kidney. The sudden occurrence of albumin in the urine of a patient who is suffering from a malady that may cause renal infarction, should always raise a suspicion of its occurrence; and it should be remembered that in morbus cordis, albuminuria and hæmaturia are very often dependent rather on infarction than on the presence of congestion of the kidney or of actual nephritis. The occurrence of sudden pyuria, in cases where septic embolism of the kidney is possible, should also be borne in mind, as an erroneous diagnosis of pyelitis, based on the presence of pus in the urine, may be made in such cases.

Prognosis.—The prognosis of renal infarction depends necessarily on its cause, and, in the vast majority of cases, it is of no great importance of itself, except as affording evidence of a grave and very often fatal underlying complaint. In septic cases the occurrence of renal infarction with the formation of renal abscess is not always very serious, but here again the diagnosis will turn generally on the nature of the original illness. Single abscess in the kidney may burst into the pelvis and be discharged without any serious result ultimately following. In other cases, however, the abscesses may rupture externally through the capsule of the kidney, and produce a perinephritic abscess, the prognosis of which will depend very largely on whether it be recognised and efficiently treated; such abscesses, if not diagnosed, may go on to a large size, and extend in various directions, more especially upwards, so as to give rise to a subphrenic abscess, or even rupture into the lung.

THROMBOSIS.

Thrombosis of the renal artery is a rare lesion, notwithstanding the fact that in many affections of the kidney, and more especially in Bright's disease, extensive disease of the coats of the arteries exists. Occasionally thrombosis results as a sequence to endarteritis, and in a few cases the thrombosis is sufficiently extensive to cause necrosis of large areas of the kidney substance, and more especially of the cortex. In some cases the necrosis following the thrombosis is of a diffuse patchy character; in others the entire cortex may undergo necrosis, similar to that seen as a result of embolism.

This condition cannot be diagnosed; the main interest of it lies in the fact that in one case, described by Lawrence and myself, where the entire cortex of both kidneys had undergone complete necrosis as a result of thrombosis of the interlobular arteries, the condition produced complete suppression of urine, lasting for seven days, with the clinical picture of latent uræmia that is characteristic of calculous obstruction. This case would support the view that ordinary uræmia is not dependent upon the mere retention of the normal urinary constituents, since, notwithstanding the complete suppression of the urinary excretion, owing to the destruction of the excretory portion of both kidneys, all the phenomena characteristic of acute uræmia were absent.

BRIGHT'S DISEASE.

Bright's disease is an inflammatory affection of the cortex of the kidney, usually progressive in type, and accompanied by secondary effects, especially on the vascular system and other parts of the body.

Richard Bright's great discovery was that certain varieties of dropsy were accompanied by albuminuria, and to the kidney lesions causing this albuminuria the term Bright's disease was applied. It was found very soon that a large number of kidney lesions, of great diversity of character, were associated with albuminuria and the various secondary phenomena that are characteristic of Bright's disease. Controversies began then, and exist to the present time, as to whether these different forms of kidney lesion are stages in one and the same malady, or whether, on the other hand, they are more or less independent and separate affections, with no greater resemblance to one another than that they have a similar etiology, and that in some respects the symptoms the different affections produce are allied.

At the present time all authorities agree in recognising acute Bright's disease and chronic Bright's disease, and, further, in recognising that chronic Bright's disease may be the sequel of acute Bright's disease, or more frequently may be chronic from the outset. There is no great discrepancy of opinion as regards acute Bright's disease; and pathologists agree that the cases possess a general resemblance to one another, the only variations being dependent on the fact that in some forms the inflammatory mischief produces the greatest damage in the glomeruli; in others in the tubules; but in all forms of acute Bright's disease the glomeruli, the tubules, and the interstitial tissues are all affected, but to varying extents. Chronic Bright's disease, however, is the form in which the greatest diversity of opinion, as to its nature and forms, exists. Formerly both the amyloid kidney and the granular kidney were included under Bright's disease. This arose from the fact that many forms of the waxy kidney present clinical characters that are distinguishable only with difficulty from Bright's disease; and, further, that in many cases of Bright's disease certain portions of the kidney have undergone amyloid degeneration. Another reason for including the amyloid kidney amongst the varieties of Bright's disease, was the fact that some specimens described originally by Bright were really

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instances of amyloid kidney, which was more common in his day than now, owing to the diminution in the frequency of amyloid disease, due mainly to modern improvements in surgical treatment, since the malady was more especially dependent on cases of bone disease associated with prolonged suppuration. The granular kidney was included by many under the term of Bright's disease, owing to the fact that in many forms of Bright's disease, especially when chronic, there is a considerable overgrowth of fibrous tissue, leading not only to toughness of the organ, but also to irregularities of the surface; and it was difficult to draw a hard-and-fast line between some forms of so-called contracted kidney, dependent on Bright's disease, and the true granular kidney associated with widespread arterial degeneration. If the term granular kidney is to be used to designate a kidney that exhibits irregularities of the surface, owing to an excessive development of fibrous tissue, then unquestionably granular kidney must be held to include some varieties of Bright's disease, and conversely Bright's disease some varieties of granular kidney. On the other hand, if the term granular kidney is to be restricted to that form of renal lesion which is associated with widespread arterial degeneration, and where the kidney lesion is only part of extensive arterial changes, it should not be included under Bright's disease, inasmuch as it is an independent affection, which only distantly resembles it. Another argument that has influenced some writers in including granular kidney amongst the varieties of Bright's disease, is the fact that in all the varieties of Bright's disease, and in the granular kidney, all the elements of the kidney are affected by the morbid processes, but to different extents; in some the tubules suffer to a greater extent; in others the interstitial tissue. Formerly it was thought that this selective action of a morbid process was greater than is now recognised. Inasmuch as in chronic Bright's disease the interstitial tissue is affected to some extent, and in the granular kidney associated with arterial sclerosis the renal tubules are also affected in a certain degree, it is difficult to draw a hard and fast line between certain forms of chronic nephritis on the one hand, and the red granular kidney on the other. So far, all these renal affections might perhaps be included in one great group, the varieties being regarded as due to the different incidence of morbid processes on the different renal elements. This view, however, is untenable, if the subject be looked at, not only from the morbid anatomy, but also from the clinical standpoint, since the etiology and symptomatology of Bright's disease, on the one hand, and of the true granular kidney on the other, present great points of difference. For these reasons, the granular or arterio-sclerotic kidney will not be included as a variety of Bright's disease, but will be looked upon as an independent affection.

Chronic Bright's disease presents great differences in different cases, both as regards its morbid anatomy and its symptomatology, and some forms of it are difficult to separate from the granular kidney.

It is customary to recognise the following varieties of chronic Bright's disease—(1) The so-called large white kidney. In this condition the kidneys are greatly enlarged, being frequently twice the normal size, and in some extreme cases the organ has been described as weighing a pound or more in weight. The older writers evidently found this form more common than we do at present, and it is probable that many of the cases of so-called large white kidney were cases in which, in addition to the lesions of Bright's disease, amyloid degeneration was also present; and in some cases, described under this heading, it is probable that the lesion was really entirely that produced by waxy disease. At present the large white kidney is not often seen in the post-mortem room. (2) The kidney most frequently found in cases of chronic Bright's disease is one where the organ is a little above the normal size, weighing some 6 or 7 oz., where it is white in colour, and presents the usual appearances described under the large white kidney, with the exception that it is not so big as those formerly described. (3) A kidney not uncommonly found in chronic Bright's disease is one where the organ is of diminished weight and size, tough, and white or mottled in colour, but presenting an exceedingly granular surface. This kidney is perhaps best described under the term of a contracted white kidney, or the small white kidney, or the pale granular kidney. (4) In rare cases a small white kidney is found, but presenting an exceedingly smooth surface. This, however, is of exceptional occurrence.

Speaking broadly, therefore, two great varieties of chronic Bright's disease may be recognised; in the first the kidney is smooth and pale, and of approximately the normal size, or slightly increased in size. This may be spoken of as the white kidney, as perhaps it does not merit, as was formerly thought, the designation of large white, as it is only large in exceptional cases. The second variety is the small, contracted, or pale granular kidney, which perhaps is best described under the name of the contracted white kidney, so as to clearly differentiate it from the true granular kidney, an independent affection in which the kidney is usually red in colour.

ACUTE BRIGHT'S DISEASE.

Etiology.—Bright's disease is generally dependent upon the action of some toxic agent on the renal structures; in many cases the toxic agent is ingested; in others it is formed in the body, as a result of pathological processes in other parts of the body; in some cases the production of toxic agents in the body is probably a result of perverted metabolism only.

Although the great majority of cases of Bright's disease are unquestionably of toxic origin, cold is still recognised as a cause of this renal lesion by a number of writers, and cases are undoubtedly seen where the renal lesion has apparently resulted from exposure to cold. The agency of cold, however, as a direct and specific cause in the production of disease, is not so widely recognised as it was formerly, *e.g.* cold as a cause of peritonitis, pleurisy, etc.; and in the case of Bright's disease, which is said to follow on exposure to cold, it is possible that in many cases there are other and toxic causes at work, and perhaps more especially alcohol. Cold was supposed, on theoretical grounds, to be a cause of Bright's disease, owing to some close relationship existing between the cutaneous vessels and the renal vessels, and it was supposed that exposure to cold, by producing constriction of the skin vessels, might produce intense congestion of the renal vessels, ultimately passing on to inflammation. Bright's disease is not uncommon in workmen and others engaged in hot shops and passing thence into the cold open air. The malady is not infrequent amongst boiler-cleaners, engineers, fitters, and other such occupations, but it is difficult in such cases to eliminate entirely the action of alcohol, lead, and other toxic causes, and it is possible that cold may act only as a predisposing cause. Similarly it has been thought that exposure to cold during the incidence of, or during the convalescence from, acute specifics might act in the same way, but here also toxic agents are at work.

Bright's disease is frequently seen as a complication or sequel of certain acute diseases, but more especially does it occur during the convalescence from specific fevers; and scarlet fever is the one that is most liable to be followed by this complication. It also occurs, but not so frequently, as a complication of typhoid fever, smallpox, measles, chicken-pox, cholera, diphtheria, rheumatism, pneumonia, and other acute diseases. It is not very uncommon as a complication of secondary syphilis, although in such cases it may be perhaps put down as due to the action of cold; but exposure to damp and cold during the incidence of secondary syphilis is not infrequently followed by the occurrence of Bright's disease.

When Bright's disease occurs as a complication of any of the febrile diseases mentioned above, it occurs usually during the latter stages of the disease or even during convalescence; and it is remarkable that this should be so, inasmuch as all these diseases, during the height of the febrile process, may be accompanied by a certain amount of nephritis, causing the well-known febrile albuminuria. Although usually occurring in the latter stages or during convalescence sometimes, and especially in scarlet fever, pneumonia, and typhoid fever, acute Bright's disease may develop during the height of the initial malady, and exceptionally this is also the case in diphtheria.

Bright's disease is occasionally seen as a complication of burns and in certain skin diseases, *e.g.* eczema, pityriasis; and it is possible that in these cases the renal lesion is really dependent upon the formation in the skin of toxic bodies and their subsequent absorption and excretion by the kidneys. A number of substances are liable to cause acute Bright's disease; the most important are cantharides, turpentine, mineral acids, carbolic acid, and certain potash salts, such as the chlorate and the bichromate; and the latter may be used in the laboratory for the purpose of producing nephritis. Alcohol, lead, and the gouty poison may also produce Bright's disease, though, in the case of these substances, the lesions produced are usually those of chronic Bright's disease.

The Bright's disease which is occasionally seen complicating pregnancy was formerly looked upon as produced mechanically by the pressure of the uterine tumour on the renal veins; it is possible, however, that in reality such cases are of toxic origin, and that the lesion is dependent on the formation and absorption of some toxic substances as yet undetermined.

Bright's disease is also seen both in its acute and chronic forms as a complication of diabetes, and here also, though its etiology is quite obscure, it is possible that it is of toxic origin. Some lesions of the kidney, certainly the granular kidney, may be produced as a sequel of excessive eating, and more especially as the result of eating large quantities of meat. It is possible that Bright's disease may arise in the same way. The occurrence of albumosuria in some cases of Bright's disease and of granular kidney may be taken as evidence of disordered metabolism, possibly of intestinal origin. Albumosuria may occur in renal disease, in the absence of inflammatory complications, and of bone disease; and its occurrence is difficult of explanation, unless it is due to derangements in the functions of digestion and of absorption.

Although toxic agencies are the most important cause of Bright's disease, there are certain predisposing causes of some importance.

Kidneys that have undergone hypertrophy are apparently especially liable to be affected by Bright's disease. This is seen in unilateral kidney, as a result of absence or atrophy in one kidney; and also in cases of bilateral hypertrophy, as in diabetes mellitus.

Occupation, as mentioned above, such as that of engineer, boiler-cleaner, etc., is a factor of some little importance in predisposing to this disease; and some occupations not only act as predisposing causes, but also as exciting causes, especially of chronic Bright's disease; and amongst this category must be included all occupations in which lead poisoning is apt to ensue, such as glaziers, potters, painters, artificial florists, etc. Indulgence in alcohol is unquestionably a predisposing cause both of acute and chronic Bright's disease, and in the latter case it may be also an exciting cause, but in acute Bright's disease it only acts as a predisposing cause.

Exposure to cold and wet is also probably frequently a predisposing cause, both in the case of alcoholics and in the convalescence from acute specifics.

Phthisis is not uncommonly complicated by Bright's disease, and here probably the mischief is dependent on toxic agencies. Bright's disease is in such cases often complicated further by the presence of amyloid degeneration.

Age has a considerable influence in predisposing to Bright's disease, and also has an influence on the incidence of the different varieties of the malady. Bright's disease is most common between the ages of 3 to 40 years, and the contracted white kidney, or small white kidney; is more frequent in young adults under 25 than in the more elderly. Bright's disease is more common in men than in women, in the proportion of 3 to 2.[1] In some cases, hereditary influences, or at any rate family influences, can be traced in the etiology of Bright's disease. The disease not only occurs sometimes in more than one member of the same family, but it is also not very uncommon for it to occur and run a rapidly fatal course in the young. Several instances have been collected of contracted white kidney, and also of granular kidney having occurred in young children as well as in several members of the same family. Another predisposing cause of some little importance is the presence of passive congestion of the kidney from cardiac disease, and in such cases the supervention of chronic or subacute Bright's disease is not uncommon.

Acute Bright's disease is most frequently seen as a sequel of acute specific fevers, or else from the combination of exposure to cold, and the action of some toxic agent, such as alcohol. In a few cases it may supervene as the result of pregnancy, and it is rarely seen as a complication of diathetic diseases, such as diabetes. Chronic Bright's disease may supervene as a sequel of the acute disorder, but in the great majority of cases this malady is chronic and insidious from the onset. In some cases the patients may suffer from acute Bright's disease, and chronic Bright's disease may subsequently develop without the one malady passing on into the other. The patient may recover from the acute disorder, and then, after an interval, suffer from a recurrence of the former attack, and this may occur several times in succession, until finally the disease becomes chronic. Although in the great majority of cases of chronic Bright's disease the malady is chronic from the onset, in a few cases the onset of symptoms is of sufficient suddenness for the malady to be described as subacute.

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[1] The granular kidney is not only more common in men than in women, but is also more frequent with advancing years, and is most commonly met with between the ages of 40 and 55.

Morbid anatomy.—In acute Bright's disease the kidneys are usually increased in size, heavy, gorged with blood, and of a dark red colour, the congestion being most marked in the stellate veins of the capsule and in the pyramids. The colour of the organ on section depends largely on the duration of the malady, and if the acute disease lasts for a month or six weeks before being fatal, the kidneys may be quite white or marbled at the time of death; the colour is very largely dependent on the degree to which degeneration has occurred in the tubular structures. The capsule strips off readily, leaving either a uniformly congested surface or more frequently a mottled or marbled surface. The cortex is swollen, of a greyish yellow colour, and opaque in appearance. The pyramids are intensely congested. The glomeruli can frequently be seen as bright red points, and streaks of hæmorrhage are not uncommon, especially in the medulla. The glomerular chamber generally contains blood corpuscles, and the vessels of the tuft have undergone hyaline degeneration, and there may be a considerable amount of exudation of inflammatory products both in the chamber and between the loops of the glomerular vessels. The epithelium lining the glomerular chamber has commonly undergone considerable proliferation, so as to form crescentic masses that are seen on section in the space between the basement membrane of the capsule and the glomerular tuft. The vessels in the capillary tuft may be thrombosed. There is commonly a considerable amount of congestion, with exudation and proliferation in the loose connective tissue surrounding the glomerular chamber, so that in the latter stages of acute nephritis the capsule undergoes considerable thickening. The changes present in the glomeruli vary in intensity in different forms of Bright's disease and in different parts of the same kidney, the distribution of the lesion in the kidney rarely being uniform. The glomerular changes are most marked in the Bright's disease arising from scarlet fever, and they are frequently spoken of as glomerular nephritis. The changes produced in the capsule interfere very materially with the circulation through it, both in the acute stage of the disease and later, when, owing to the formation of fibrous tissue, the glomerular tuft becomes compressed and its vessels more or less strangled. The obstruction to the circulation in the glomeruli may in part account for the changes which are found in the epithelium of the convoluted tubules, inasmuch as the circulation through the glomeruli cannot be interfered with without seriously damaging the nutrition of the convoluted tubules, since the veins passing from the glomerulus supply the convoluted tubules with blood.

The tubules of the cortex of the kidney are dilated, and in many places the epithelium is shed to varying extents; where the epithelium remains, the cells have undergone marked changes, having lost their striated border, and in many cases they have become hyaline or glass-like in appearance, retaining, however, their nuclei. In addition to this hyaline degeneration, the cells may show cloudy swelling and the signs of fatty degeneration. In the more severe forms of acute Bright's disease, considerable portions of the tubules may be destitute of epithelium. The lumen of the tubules is occupied by blood corpuscles, disintegrated renal cells, and casts, which may consist simply of coagulated blood plasma, forming hyaline and granular casts, or the casts may consist of aggregations of renal cells which have undergone more or less extensive degeneration.

The dilatation of the tubules is dependent on the blocking caused by these shed products, and the urinary secretion pent up behind this obstruction produces the dilatation. The enlargement of the organ is in part dependent on the dilatation and distension of the convoluted tubules.

The changes in the medulla are of a similar character, but are not usually so marked in extent. The interstitial tissue presents the appearances due to vascular engorgement and œdema, and these cause a separation of the tubules. In many cases there are hæmorrhages of varying size dependent on the acute inflammation, and in addition small-celled infiltration. All the above changes may be distributed uniformly throughout the kidney, or more frequently they are developed to varying extents in different parts of the organ. In some varieties of Bright's disease and of nephritis the round-cell infiltration of the interstitial tissue is a marked feature, and, owing to the accumulation of the leucocytes in disseminated masses, an appearance simulating that of multiple new growths is produced. To this variety the name of lymphomatous nephritis has been applied, and this is more especially seen in some forms of nephritis following typhoid fever.

The inflammatory changes present in the interstitial tissue are especially marked round the glomerular chambers and also round the arteries, and the periarteritis is not uncommonly complicated further by the presence of varying amounts of endarteritis causing considerable thickening of the sub-endothelial coat and so obstructing the lumen of the vessels.

Symptoms.—The onset of acute Bright's disease is usually sudden, and in many cases the first symptom is the occurrence of dropsy. In others, however, the malady is ushered in by more general symptoms, such as headache, vomiting, and pain in the back. Pain is not usually a prominent symptom. The dropsy is most marked in the subcutaneous tissue, and probably it is most characteristic for it to occur in the scrotum and vulva, though in other cases it may first be noticed in the eyelids, and in others in the legs. The distribution of the dropsy, however, depends in part on the degree of illness at the onset; if the person is able to get about, the dropsy will be most marked in the legs; on the other hand, if they are confined to bed, it will be found most marked in the face and over the sacrum, and this is the explanation of the well-known fact that renal dropsy may be most marked in the face in the morning and in the legs in the evening. The dropsy sometimes involves almost at once the whole of the subcutaneous tissues.

In some of the more severe forms of acute Bright's disease, occurring after scarlet fever and other acute specific diseases, pyrexia may be present. The fever as a rule is moderate in amount, not rising above 102° or 103°; but in the vast majority of cases of even acute Bright's disease the temperature may not rise higher than 100°. In a small proportion of cases rigors and high fever may be present.

In many cases of acute Bright's disease, the first symptoms to attract attention to the morbid process are not the general ones, but the changes which are produced in the urine. This is scanty, and it may even be suppressed, although it is more usual for the suppression to be partial: but the onset of the disease may be marked by the patient suddenly passing a few ounces of dense, high-coloured, blood-stained urine. The urine is usually smoky in appearance, but in some cases at the onset of the disease a considerable quantity of blood is passed, imparting a uniform red colour to the fluid. In others it is dark, and may even be porter-coloured. A copious deposit of casts and blood corpuscles may occur, and albumin is present in large quantity. In some cases of acute Bright's disease, the urine may present all the changes characteristic of a violent nephritis, and the patient show little or no dropsy. In others, dropsy may be a prominent symptom, and the urine may be only little more than smoky, although it will contain a large quantity of albumin. The excretion of urea is diminished in acute Bright's disease, although the percentage excreted may be high. The diminution of urea, however, is due to several causes, and not entirely to the inefficiency of the kidney as an excretory organ. The appetite is poor, nausea and vomiting are often prominent symptoms, so that what little food is taken is in part rejected, and this will account to a certain extent for the considerable diminution in the urea excretion.

Uræmic symptoms are present to varying extents in different cases of acute Bright's disease, nausea and vomiting being almost always present. In the more serious forms of acute Bright's disease, accompanied by suppression of urine, the more severe uræmic symptoms, such as twitchings, amaurosis, epileptiform seizures, dyspnœa, drowsiness, and coma are frequently present. In the form of the malady that follows scarlet fever, suppression of urine and uræmia are prominent symptoms. Death in acute Bright's disease is frequently due to uræmia, and more especially to the form associated with coma and convulsions. In the variety which is supposed to be due to exposure to cold, dropsy is a more prominent symptom, and indeed, in post-scarlatinal Bright's disease, even when severe, dropsy may be absent.

Anæmia is always a marked symptom, the patient's mucous membranes becoming exceedingly pale, and the face assuming an opaque, pasty appearance. The skin of the body generally is dry, and it is often difficult to promote sweating. Hæmorrhage may occur from the nose and from the gums, stomatitis with a scurvy-like condition of the gums being a not uncommon phenomenon. The dropsy may not in severe cases be limited to the subcutaneous tissue, but may also affect the serous cavities. It is said that in some cases the dropsy in the serous cavities may be extreme in amount, and that in the subcutaneous tissue quite limited. The pulse in acute Bright's disease may show signs of increasing tension, and in the course of a short time, *e.g.* three to six weeks, the heart may give evidence of definite enlargement from hypertrophy. Cardiac dilatation may supervene earlier than this in the course of the malady. Retinal changes are rare in acute Bright's disease, but papillitis and hæmorrhages and even retinitis may occur. So-called "secondary inflammations," such as pleurisy, pneumonia, and pericarditis, are common. Skin eruptions of a papular, erythematous, vesicular, or bullous type may occur associated with uræmia.

Diagnosis.—The diagnosis of acute Bright's disease is usually easy, from the combination of several symptoms, such as the presence of dropsy, together with the signs of acute inflammation of the kidney, such as blood, albumin, and casts in the urine. The principal difficulties which present themselves are to distinguish acute Bright's disease from transitory nephritis, from infarction in the kidney, and from certain cases of passive congestion. The presence of albumin and traces of blood in the urine during the height of a febrile illness, such as scarlet fever, do not necessarily point to the onset of acute Bright's disease; they may be dependent on a transitory nephritis; and although it is difficult to draw a hard and fast line between nephritis and Bright's disease, still there are points of difference, and the most important of all is the fact that whereas a nephritis may be transitory and clear up entirely, the outlook in acute Bright's disease is more unfavourable. In the transitory form of nephritis from acute specific diseases, dropsy is absent; and although albumin is usually present, blood is either absent from the urine or else there is only a mere trace. In acute Bright's disease, on the other hand, dropsy is usually a prominent symptom, and blood is often present in large quantity.

Hæmorrhagic infarction is liable to occur in a number of conditions, more especially in cardiac diseases, such as simple and especially malignant endocarditis; and its onset may be marked by the occurrence of pain and the passage of considerable quantities of blood and albumin in the urine. All these effects are produced with great suddenness, and are of very transitory duration, and are not accompanied by dropsy. The mistake of confounding hæmorrhagic infarction with acute Bright's disease is only liable to occur if undue stress is laid on the mere presence of blood, together with considerable quantities of albumin in the urine.

In passive congestion of the kidney, arising from heart disease, dropsy of a cardiac type may be present, blood and albumin may also be present in the urine, and the urine may be very considerably diminished in quantity. In such cases it may often be difficult to determine with certainty whether Bright's disease be present or not. If passive congestion alone be present, although the urine be albuminous and may contain blood, casts of definite renal elements will not be present although hyaline and blood casts may be abundant.

Difficulties may arise in the diagnosis of acute Bright's disease, in rare cases where dropsy is a prominent symptom, and where not only blood is absent from the urine, but where there may be little, or even it is said no albumin present. This, however, is rare, and the absence of albumin, if it occurs, is only temporary.

It must be remembered that in some forms of serious Bright's disease the first symptoms manifested may be those of uræmia, such as elipeptiform seizures. This may occur, more especially in the Bright's disease which is sometimes associated with eclampsia; and if the urine is not examined, the uræmic nature of the seizures may not be recognised.

Prognosis.—The prognosis of acute Bright's disease depends very largely on its cause; that complicating scarlet fever or diphtheria is more fatal than that which is said to occur as a result of cold. Unfavourable symptoms are the persistence of dropsy, and the gradual increase in the anæmia, and the presence of severe uræmic symptoms. Acute uræmia, however, in acute Bright's disease, is not so serious as acute uræmia in chronic Bright's disease.

The first sign of improvement in acute Bright's disease is usually an increase in the quantity of urine secreted, and soon the dropsy begins to subside. In a large proportion of cases the dropsy will entirely disappear, and later the albumin will diminish, and ultimately may also disappear. It is not uncommon, however, even in cases which ultimately recover, for recurrence both of the dropsy and of the albuminuria to take place. In a small proportion of cases, the condition, however, becomes chronic, and ultimately proves fatal after some months, neither the dropsy nor the albuminuria undergoing diminution.

Treatment.—Patients suffering from acute Bright's disease should be put to bed, and must remain in bed until all the acute symptoms have disappeared, inasmuch as recrudescence of the malady is extremely prone to occur as the result of injudicious exposure to cold.

The diet in acute Bright's disease should be restricted to milk, barley-water, and gruels. Meat extracts, beef-tea, chicken-broth, etc., are of very small nutritive value, and are decidedly injurious in the case of renal disease, owing to the presence of quantities of nitrogenous extractives in these so-called foods, which therefore increase the work of the kidney.

In the acute stages of the disease probably a very limited diet, such as 1 to 2 pints of milk in the twenty-four hours, is sufficient, although of course such a restricted diet as this cannot be continued for any length of time; 3 to 4 pints of milk is the minimum amount of milk necessary for the dietary of an adult. In the acute stage of the disease, where it is very necessary to spare the kidney as much as possible, and where the appetite is always poor, it is sufficient to give such a restricted diet as 1 or 2 pints of milk, freely diluted with barley-water or soda-water.

It is usually recommended that the patient should drink freely of simple mineral waters or alkaline waters or lemonade, to relieve thirst. Stimulants should be avoided. Sweating should be encouraged, although it is often difficult to produce any marked effect on the skin, owing to the remarkable manner in which the secretion of sweat is arrested in many cases during the disease.[1] Hot drinks and hot-air baths may be prescribed for this purpose, and the patient should be warmly covered in bed between blankets. If pain in the back is a prominent symptom, it may be relieved by hot applications or by cupping.

As in most cases the secretion of urine is very seriously diminished in amount, measures must be used to promote the activity of the skin, and in addition to those mentioned above, jaborandi or pilocarpine may be ordered. These drugs may often be usefully combined with the hot-air bath. If the latter be used, it should be employed with caution at first, and it is not advisable to have a temperature higher than 120° F. on the first occasion, although subsequently the bath may be raised to a temperature of 170° F. or 180° F. If the hot-air bath produces symptoms of cardiac weakness, very good results can often be obtained by a simple hot-water bath. Jaborandi and pilocarpine are often very successful agents for causing sweating, but in some cases they cause a considerable amount of nausea and abdominal disturbance, and they may produce very serious symptoms of collapse, from the action of the drug on the heart, so that they should be employed with caution. The bowels should be kept freely open with saline purges, such as sulphate of soda and sulphate of magnesia; and inasmuch as it is desirable to promote free watery evacuations, it is best to give these substances in a concentrated form. Jalap is also useful; excessive purgation must, however, be avoided, as enteritis is not an uncommon complication of Bright's disease; and a profuse diarrhœa may be started by injudicious purgation, which cannot be readily prevented, and which may cause increased exhaustion.

Uræmia must be treated by measures directed to increase the activity of the skin and bowels. If these fail, as is not uncommonly the case, venesection is useful, and saline transfusion after venesection is often of great value. Chloroform may be used to mitigate the severity of the convulsions, and morphine may also be used for this purpose, but many regard its use as dangerous. It is more especially of value where convulsions are present.

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[1] This fact, together with the action of cold in predisposing or exciting Bright's disease, has led to the view that a toxic action might be exerted on the kidney by the retained products that should be excreted by the skin.

The dropsy must be treated by measures promoting the increased activity of the skin and the removal of fluid by the bowel; but if these measures fail, and the dropsy increases to such an extent as to become directly dangerous to life, it may be necessary to puncture the limbs with Southey's tubes, or aspirate the serous cavities, or even to make incisions in the legs. Great care must be taken in the last case to maintain strict antiseptic precautions, as the tissues of patients suffering from both acute and chronic Bright's disease have a lowered resistance, and hence septic complications, often of a very dangerous character, are liable to ensue.

Vomiting in Bright's disease must be treated with bismuth, hydrocyanic acid, ice, and small quantities of food. If cardiac weakness from dilatation ensues, it should be treated with digitalis and strychnine. Authorities differ with reference to the question as to whether diuretics should be used in acute Bright's disease. On theoretical grounds much can be said against their use, but clinically it cannot be urged that any marked evil results have been known to follow their employment. Citrate of potash, digitalis, caffeine, are the best to employ; but the last, although a most powerful diuretic, very soon loses its effect, and it is not uncommon for all diuretics to fail in Bright's disease. In all cases, however, water should be freely taken, as it is itself a diuretic. No drug is known to produce any effect on albuminuria during the acute stage, but during the subsidence of the malady arsenic is often of use, probably by its action in improving the general health.

During convalescence the anæmia should be treated by the free administration of iron, and the diet should gradually be increased. Patients who have suffered from acute Bright's disease should after convalescence go to a warm and equable climate, and it may be necessary for them to winter abroad in such countries as Egypt, Algiers, or Madeira.

CHRONIC BRIGHT'S DISEASE.

Etiology.—The chronic disease can often be traced to the effects of some slow-acting toxic agent, such as lead, alcohol, or gout, and perhaps also to exposure to cold.

The onset of the contracted white kidney, a disease always chronic in its progress, is usually insidious, and the course of the malady so slow that its causation often cannot be traced; but some writers have held the view that this form of Bright's disease is really a terminal stage of certain varieties of acute Bright's disease, and of the other form of chronic Bright's disease. They regard the contracted white kidney as a later stage of the so-called large white kidney, much in the same way as atrophic cirrhosis of the liver is regarded by some as a sequel of certain forms of hypertrophic cirrhosis. According to this view, acute Bright's disease might terminate with the formation of a contracted kidney, the large white kidney forming an intermediate stage in its evolution. An antecedent history of scarlatinal nephritis can be obtained in some cases that come under observation with, and are fatal from, a contracted white kidney, but such a history in such cases is exceptional. The great bulk of cases of contracted white kidney certainly give no history pointing to the previous existence of the large white kidney, and these patients have not, in my experience, suffered formerly from the dropsy which is so characteristic an effect of the large white kidney. The contracted white kidney may perhaps arise as a remote sequel of a former attack of acute nephritis, but it probably is not to be looked upon as a mere final atrophic stage of chronic Bright's disease, but as an independent variety which frequently begins insidiously as such.

Morbid anatomy.—The appearances of the kidney in **chronic Bright's** disease present very great differences in different cases, both to the naked eye and in the microscopic appearances, and it is difficult, if not impossible, to gauge accurately the microscopic changes from the naked-eye appearance of the organ. The most common variety is that formerly described as the large white kidney, although, as has been already mentioned, the description *large* is scarcely applicable at the present day to the most common instances of it. In the most frequent examples of this form of the disease, the kidneys may be increased to the extent of weighing 6 or 7 oz. In exceptional cases the increase in the size of the kidney is greater, and instances have been recorded of the weight reaching 1 lb. The surface of the organ is white or marbled; the stellate veins are conspicuous; the capsule is thin, leaving on stripping a smooth surface; the cortex is thickened, and is usually of a peculiar opaque yellowish white colour; the pyramids are congested. The glomeruli, the tubules, and the interstitial tissue all show evidence of the morbid process, but to different extents in different cases. Usually the glomeruli are large, and their capsule is thickened. There is a certain amount of cellular infiltration of the glomerular tuft, and the epithelial lining in the glomerular chamber has undergone proliferation, and in some cases great masses of cells are found in this position occupying the space between the basement membrane and the capillaries of the tuft. The walls of the capillaries show signs of hyaline degeneration.

The tubules of the cortex are dilated, and in many places the epithelium is shed; in others it remains, presenting fairly normal characters; and in others the cells composing it have undergone hyaline degeneration. In many places the epithelium has undergone fatty degeneration. The tubules generally contain casts.

The interstitial tissue is in all cases affected, and presents either the appearance of young cell infiltration or overgrowth of fibrous tissue to a varying extent; the amount of this will depend largely on the duration of the disease, peri- and endarteritis are also present.

In the other form of chronic Bright's disease—the contracted white kidney—the organ is small, weighing some 2 or 3 oz., the capsule is thickened, and on stripping leaves an exceedingly granular surface, although its removal may not tear the renal substance to the extent that is seen in the true granular kidney. The cortex is greatly diminished in width, the distinction between it and the medulla is obscure, it is pale and opaque in colour. The glomeruli are small, greatly thickened from the formation of fibrous tissue, and the glomerular tuft compressed and atrophied. The tubules have in great part lost their epithelium, and where it remains it shows well-marked signs of hyaline degeneration. In many parts of the kidney no remains of the convoluted tubules can be found, and here the organ consists of nothing but masses of fibrous tissue. The interstitial tissue of the kidney is everywhere greatly increased in amount, and in many places nothing but atrophied glomeruli embedded in these masses of fibrous tissue can be found. Here and there certain of the tubules may be seen lined with more or less normal epithelium, and in some cases dilated tubules are seen. The diminution in the size of the organ is mainly dependent on the shrinking and disappearance of a large number of the tubules, on the atrophy and compression of the glomeruli, and on the contraction caused by the overgrowth of the interstitial fibrous tissue.

Microscopic appearances of this variety of Bright's disease present considerable resemblances to those seen in the granular kidney, and in both conditions the renal arteries are usually thickened, and present marked signs of peri- and of endarteritis.

Symptoms.—In a certain proportion of cases acute Bright's disease passes on by insensible gradations into chronic Bright's disease, but in a larger number of cases chronic Bright's disease begins insidiously.

The initial symptoms present great variety, but the most constant are progressive failure of strength, and very commonly considerable wasting; in a few cases no other marked symptoms are observed at first. The wasting in some cases, and especially in that form of the disease due to the contracted white kidney, may be so great as to simulate some varieties of malignant disease. In a large number of instances, the first symptom that attracts attention is the occurrence of dropsy. In others, uræmic symptoms of slight severity, such as nausea and vomiting, occur. Many patients with chronic renal disease seek advice on account of severe headache, and sometimes for impairment of vision, dependent on the presence of retinal hæmorrhages and albuminuric retinitis.

It is unusual for persons with chronic Bright's disease to seek advice primarily on account of the remote cardiac effects caused by the high tension; this is more usually the case in the granular kidney. When chronic Bright's disease is fully established, cardiac symptoms such as palpitation and breathlessness are prominent, and many such patients suffer from all the symptoms characteristic of valvular disease, more especially of mitral regurgitation.

The urine in chronic Bright's disease presents considerable differences, according to the form the malady assumes, and according to whether dropsy is present or not. If dropsy be present, the urine is usually scanty in amount, 20 oz. or less are secreted per diem. It is pale in colour, and contains a large but variable quantity of albumin, sometimes as much as 5 per cent., in which case it will become solid on boiling; in other cases not more than 1 or 2 per cent. is present, and in cases undergoing improvement even less than this. Casts are present in abundance, granular, hyaline, and epithelial, and they frequently show signs of degeneration. The specific gravity of the urine varies, and it is usually between 1010 and 1020. It is uncommon for patients with advanced Bright's disease to pass a urine with high specific gravity, even when dropsy is present.

In the form of Bright's disease associated with the contracted white kidney, and where dropsy is usually absent, the urine is pale and more abundant in quantity, the amount passed being often greater than normal, and amounting to 60 or even 100 oz. per diem. The specific gravity is low, 1010 to 1015, and the amount of albumin, although not so large as that seen in the other variety of the disease, is yet considerable, amounting perhaps to one-sixth or one-third. Although the percentage amount of albumin in these cases is usually smaller than in the other form of Bright's disease, yet, owing to the quantity of urine passed, such patients may lose a greater amount of albumin in the twenty-four hours than in the other variety of the disease.

In this variety of chronic Bright's disease it is not unusual for 20 to 40 grms. of albumin to be lost in the urine per diem.

It is difficult to separate with accuracy the symptoms characteristic of those two forms of chronic Bright's disease. Dropsy is a more characteristic feature of the first variety than of the second, and patients suffering from the contracted white kidney are most likely to come under observation, suffering from vague symptoms of ill health, such as wasting, headache, and loss of strength. It is remarkable that the great majority of such patients neither suffer at the time from dropsy, nor do they give a history of having suffered from dropsy. It is probable that the two forms of the disease are really independent, although, from the fact that occasionally such patients give a history of dropsy, it has been supposed that the contracted white kidney supervenes, after the lapse of months or years on the other kidney lesion. Many of the patients with contracted white kidney are young, being under 25 years of age. The two types can be separated clinically, therefore, as well as pathologically. In the one form the patient suffers mainly from dropsy, together with anæmia and secondary inflammations, and in the other from a serious disturbance of nutrition, absence of dropsy, and a very great tendency to the development of uræmia.

In most cases of chronic Bright's disease the cardio-vascular system shows signs of being involved, and usually the heart shows clinically definite signs of enlargement. In many cases, however, it is difficult to detect the cardiac hypertrophy, notwithstanding its presence, owing to the coexistence of emphysema. The greatest amount of cardiac hypertrophy occurs in the cases of the contracted white kidney. The pulse tension is raised, and the arteries that are accessible to examination usually show evidence of distinct thickening. The retinal vessels present these changes to a marked extent, and they can be seen on ophthalmoscopic examination to have a wiry appearance, and this change in the arteries very often precedes the development of albuminuric retinitis and papillitis.

Papillitis, retinal hæmorrhages, and albuminuric retinitis are frequently found in all forms of chronic Bright's disease. In some cases the changes are limited to the vicinity of the macula; in others they are widespread, large masses of exudation occupying the fundus; which also contains scattered flame-shaped hæmorrhages. Although in the majority of cases the changes in the retina are more marked than those in the papilla, exceptional cases of marked papillitis are seen, causing a resemblance between the retinal changes present in renal disease and those associated with increased intracranial tension, as for instance tumour cerebri. In some cases errors of diagnosis may arise, owing to the presence of headache and vomiting in both conditions. Although the retinal changes may occur in all forms of chronic Bright's disease, they are especially marked in the form associated with the contracted white kidney, and frequently the changes in the fundus, in a person seeking advice for apparently trivial and obscure symptoms, leads to the recognition of the very grave renal lesion present.

Hæmorrhage from various mucous membranes, or, more exactly, a condition of purpura, is not uncommon in chronic Bright's disease. Hæmorrhage from the gums, from the nose, and purpuric extravasations in the skin, especially of the legs, are the most common manifestations. In some cases profuse hæmorrhage occurs into the urine, and it is probable that in some of these cases the hæmorrhage is really derived, as it is in purpura hæmorrhagica, from the mucous membrane of the renal pelvis. In some cases of contracted white kidney, the hæmorrhage in the urine is so marked, and the general wasting of the patient is so considerable, that the erroneous diagnosis of malignant disease of the kidney has been made.

The dropsy of chronic Bright's disease is not only remarkable for its amount but also for its persistence. It may undergo variations in amount from time to time, and it may indeed entirely disappear. Usually, however, it is a marked feature, and persists to a greater or less extent throughout the duration of the illness, although, as already mentioned in the case of contracted white kidney, it may be absent throughout the course of the disease. Whenever the dropsy is at all extensive, it involves not only the subcutaneous tissue, but also the serous cavities, more especially the pleuræ.

The anæmia of chronic Bright's disease is very marked, and may be so extreme as to produce an almost bloodless condition of the mucous membranes, such as the conjunctiva, lips, etc.

Gastric symptoms are usually present, nausea being especially severe. Vomiting, particularly in the morning, is also a prominent symptom. Hiccough and flatulent distension are also well marked. Diarrhœa is occasionally present, and in some cases is exceedingly severe, and in a small proportion of cases is associated with ulceration of the small and large intestine, producing a condition resembling that of ulcerative colitis. The gastritis and enteritis seen in this disease may be in part dependent on the excretion by the alimentary canal of urea and other bodies, which are present in the blood in abnormal amount.

Headache is a prominent symptom, and is often dependent on the vascular changes induced by the disease, and is therefore most marked in cases where high tension is most developed. It is frequently relieved by the occurrence of spontaneous hæmorrhage, for instance, epistaxis.

Uræmia is a prominent and formidable complication of chronic Bright's disease, and in the more severe forms of the disease it is always present to a certain extent. In the so-called large white kidney associated with dropsy, the uræmic symptoms are usually subacute or chronic, and acute uræmic manifestations are only seen when the disease is so severe as to cause almost complete suppression of urine. Chronic and subacute uræmia produces symptoms of a gastro-intestinal type, more especially nausea, vomiting, and hiccough. Other symptoms of a so-called nervous type are also present, such as twitchings of the muscles, painful cramps, and Cheyne-Stokes' respiration. The temperature of patients suffering from chronic uræmia is usually subnormal, and varies from 95° to 97°.

Where the progress of the malady is unfavourable, other uræmic symptoms of a more acute character are liable to supervene, such as drowsiness, deepening into coma, epileptic seizures, amaurosis, or dyspnœa. Uræmia, however, more particularly in its acute forms, is especially characteristic of the contracted white kidney, and here not only are the uræmic symptoms more severe but they are also very frequently of extremely sudden onset; and it is not uncommon after a vague period of ill health, often not sufficient to interfere with the occupations of the patient, for violent uræmic complications to develop, more especially delirium, mania, coma, epileptiform seizures, and hurried respiration. In such cases the quantity of urine is not always seriously diminished, and patients with this form of chronic

Bright's disease may die from acute uræmia, passing quantities of urine little below those usually seen in health; owing, however, to the dilute character of the urine secreted in these cases, the quantity of solid ingredients excreted may be seriously below the normal. It is remarkable and important to recognise, moreover, that acute fatal uræmia, associated with the contracted white kidney, may occur at a time when the patient is passing considerable quantities of urine containing amounts of urea quite comparable with those secreted in health.

Patients with chronic Bright's disease may not only succumb to uræmia, and to the mechanical effects produced by the dropsical effusions, but in addition to these, secondary inflammations of the serous cavities, pericardium, pleura, and peritoneum are common, and pericarditis is especially prevalent and fatal.

Cerebral hæmorrhage is also not uncommon, and although a more characteristic complication of the true granular kidney, it may occur in chronic Bright's disease, and it may even occur as a complication of the large white kidney. Cerebral hæmorrhage in chronic Bright's disease is necessarily a most serious and fatal complication.

Diffuse dark pigmentation of the skin is sometimes seen in chronic Bright's disease, resembling that seen in Addison's disease, but not presenting the characteristic distribution of the latter. Skin rashes also are not uncommon, such as erythema of various kinds, urticaria, eczema, pityriasis rubra, pruritus, purpura. In uræmia a papular erythema is occasionally seen, and in rare cases a bullous eruption.

Diagnosis.—The diagnosis of chronic Bright's disease is usually easy, more especially in the forms associated with dropsy. Where dropsy is absent, serious errors may arise in cases of patients presenting vague symptoms, such as wasting, if the urine be not examined. If this be neglected, and if headache and vomiting are prominent symptoms, the disease may be confounded with cerebral tumour. This is a mistake that is occasionally made, and perhaps more especially so when, during the course of the disease, cerebral hæmorrhage, causing hemiplegia or some other form of palsy, has been produced.

Uræmic seizures with the contracted white kidney, occurring suddenly, in conditions of approximately good health, may be also confounded with epilepsy, if it be not recognised that the contracted white kidney is liable to advance to a high degree without producing obvious symptoms. In all such cases, errors of diagnosis can be at once avoided by a proper examination of the urine, and of the fundus oculi.

Some cases of so-called physiological albuminuria are difficult to separate from the slighter forms of chronic Bright's disease, and here the differential diagnosis must be made on the absence of signs pointing to affection of the cardio-vascular system, on the examination of the fundus, and on the fact that in so-called physiological albuminuria the amount lost is usually small and variable, being greatly influenced by diet.

In some cases of chronic Bright's disease, marked pigmentation of the skin occurs, and I have known the contracted white kidney to be erroneously diagnosed as Addison's disease.

Prognosis.—The prognosis of chronic Bright's disease is always unfavourable, and the duration of life exceedingly uncertain, as patients whose general health is not as yet impaired by the renal lesion may die suddenly at any moment from cerebral hæmorrhage. Although this complication is most liable to occur where the signs of high tension are very marked, yet the state of the cerebral vessels cannot be gauged accurately by the examination of the heart and pulse. All that can be said is, that where high tension is obviously developed, cerebral hæmorrhage may occur at any moment, even when the general health of the patient is apparently good. Another grave difficulty in giving a prognosis, lies in the fact that not only may cerebral hæmorrhage develop at any moment, but acute uræmia sometimes develops with great suddenness, just as acetonæmia does in diabetes. Cases of acute fatal uræmia are not very uncommon in chronic Bright's disease unaccompanied by dropsy, where the general health of the patient has not materially suffered. On the other hand, patients suffering from chronic Bright's disease with intense anæmia, and passing large quantities of albumin in their urine, may live for many years.

It is usually stated that where the albuminuria and other symptoms have lasted for more than a year, recovery never occurs; this, however, is doubtful. The presence of albuminuric retinitis is also of grave import, and it is usual to state that patients suffering from this complication do not survive more than a year. This, however, is also liable to exceptions, and cases are known where patients have lived for five, seven, or even more years after the development of albuminuric retinitis. These, however, are quite exceptional cases; and the presence of albuminuric retinitis materially influences the gravity of the prognosis.

Treatment.—The treatment of chronic Bright's disease involves many questions; if the disease is of recent origin, and dropsy is a marked symptom, the patient should be kept in bed on a milk diet for at any rate some weeks, and more especially if, under this treatment, there are signs of improvement. If, notwithstanding the rest in bed and the milk diet, the albuminuria and the dropsy persist, it is probable that it is not advisable to keep adults for an indefinite period on a milk diet, and that better results are to be obtained by increasing the diet gradually to a more liberal scale. The presence of uræmic complications should always indicate the use of a milk diet. In the absence of this complication, the mere presence of chronic Bright's disease is not sufficient indication for a rigid milk diet to be maintained, as was formerly the practice, for months, inasmuch as it is impossible for an adult to maintain a satisfactory state of nutrition on a rigid milk diet. All concentrated and rich foods should be forbidden in all cases of chronic Bright's disease, and the patient should eat sparingly of meat. Vegetables, with the exception of asparagus, rhubarb, and sorrel, and most fruits, are admissible. Alcohol should be avoided either entirely, or taken in very small quantities.

Dropsy should be treated by hot-air baths in moderation, by purgation, and if necessary, as a final resort, by incision, great care being taken to avoid septic complications. The anæmia must be treated with iron and arsenic, uræmia by hot-air baths, purgation, pilocarpine; and in certain cases it may be advisable to treat uræmic convulsions by the administration of morphine. Morphine, however, is dangerous in some cases of chronic Bright's disease, where the quantity of urine is seriously diminished in amount; the mere presence of albumin in the urine, however, is not a contra-indication to the administration of the drug.

GRANULAR KIDNEY.

Syn., Cirrhotic, Contracting, or Gouty Kidney.

This consists in a degeneration of the epithelial structures of the kidney, accompanied by a great over-growth of the interstitial fibrous tissue, together with arterial degeneration, specially marked in the renal vessels, but affecting also those of the body at large to a varying extent. The true granular or raspberry kidney does not include that form described above as a contracted white kidney. Although there are many points of resemblance in the anatomy of the kidney in the two diseases, the clinical history in the two affections is quite distinct. The true granular kidney may arise either as an independent primary affection, or else as part of a widespread arterial degeneration. In the latter case it forms part of the lesions which were described by Gull and Sutton as the result of arterio-capillary fibrosis. In the cases in which it is an accompaniment of extensive arterio-capillary fibrosis, the renal lesions, although well marked, have not usually advanced to such an extent as they have in the primary form of the disease. Granular kidney in both its forms is an exceedingly chronic and insidious malady, which does not produce symptoms until it is far advanced. In many cases, indeed, it is latent throughout its course, and only the secondary complications of the disease, such as cerebral hæmorrhage, reveal its existence.

Etiology.—In the cases where the affection is part of a widespread arterial degeneration, the etiology is really that of degeneration of the arteries. In this hereditary influences play a part, and such generally acting causes as alcoholism, syphilis, laborious occupations, strains, etc. Age and sex are also important, as the disease is one that is far more prevalent in men, more especially after 40 years of age, being very rare under 25, and not common between 25 and 40. In the primary form of the disease it is probably generally the result of a chronic toxæmia, as for instance lead. Gout and lithæmia are also said to be causes. Alcohol is probably also a cause, but it is perhaps not such an effective agent in producing this malady as it is in causing chronic Bright's disease. Large eaters, and especially those who indulge excessively in meat, are said to be liable to this disease, and it is, generally speaking, a malady that is associated with so-called "high living." Hereditary influences also play an important part, and this is most strikingly seen in those rare instances in which several members of a family are affected by this disease, even in youth. Mental stress and anxiety are undoubtedly causes of the disease, and it is not uncommonly seen in men of vigorous physique, who have, from a variety of causes, been exposed to great anxiety and stress. The variety of granular kidney associated with extensive arterial degeneration is often seen as a complication in other chronic diseases, *e.g.* myxœdema, and also in various forms of insanity, so that it is a common affection amongst the inmates of lunatic asylums.

Morbid anatomy.—The surface of the kidney, on stripping the capsule, is always granular, and the removal of the capsule is always followed by tearing of the renal substance. The degree, however, of this varies necessarily in different cases. The size of the kidney is also very variable, and, speaking broadly, in the arterial form of the malady the kidneys are not so greatly diminished in size as they are in the primary form. In the latter it is not uncommon for the weight to be reduced to 2 oz., although from 2 to 3 oz. is the more common condition. The surface of the organ varies considerably in colour; the most typical variety, known as the raspberry kidney, is of a deep red colour, and the granulations are exceedingly well developed. In others the surface may be mottled, and in some rather pale; and it is for this reason perhaps, amongst others, that it is sometimes difficult to separate the naked-eye appearances of the granular kidney from some varieties of the contracted white kidney. The cortex is greatly diminished in thickness, measuring sometimes not more than $\frac{1}{16}$ in., and the medulla is deeply congested, and the fat in the hilum well marked. In some cases even it would seem that the amount of fat is increased.

Cysts, generally small in size, from a pin's-head to a split pea, are not uncommon in the cortex; sometimes they are larger, and in exceptional cases they may be present in considerable numbers. These cysts usually contain an amber-coloured albuminous fluid, containing a small proportion of urea, and not uncommonly the fluid is blood-stained.

On section the kidney is tough, and the cortex is mottled. The vessels stand out prominently, their walls being greatly thickened, and in the larger vessels it can often be seen that the thickening only occupies a portion of the circumference, so that on section the lumen of the vessel is crescentic.

There is an enormous overgrowth of connective tissue in the kidney generally, but it is not usually uniform; hence some portions of the kidney seem to be little more than masses of fibrous tissue, all the secreting and epithelial elements having disappeared. The overgrowth of fibrous tissue is most marked in the cortex, and more especially in that portion of the cortex intervening between the medullary rays. Fibrous tissue is most dense round the glomeruli, and many of these are completely obliterated, having undergone atrophy; the glomeruli remaining have dense fibrous capsules enclosing the remains of the capillary tufts, which have undergone hyaline degeneration. In addition, the capillary tuft is destroyed, owing to the overgrowth of fibrous tissue in it, and it may be said that the changes in the glomeruli are dependent on three factors—the thickening of the glomerular capsule, the compression and the destruction of the glomerular tuft by the overgrowth of fibrous tissue in it, and the hyaline degeneration of its walls; and, further, the lesions in the vessels causing endarteritis are such as to interfere mechanically with the circulation of the blood through the capillary loop. Here and there comparatively normal glomeruli are to be found, in which only the glomerular tuft is seen to be thickened, together with some cellular and fibrous overgrowth between the loops of the capillary tufts. One of the striking peculiarities of the affection is the different extents to which the glomeruli are affected in different parts of the kidney.

The tubules show a variety of changes, in many parts they are completely replaced by fibrous tissue, the cells having disappeared, and the overgrowth of interstitial connective tissue evidently flattening and collapsing the tubules destitute of their cells. In other parts, and especially in the cortex, the tubules are dilated, and, as mentioned above, in some parts they form actual cysts. In the dilated tubules the epithelium has lost its characteristic peculiarities, and the cells lining the tubules are hyaline and glass-like. In other parts, the epithelium has undergone granular and fatty degeneration, and the lumen of the tubules may contain casts. Large numbers of tubules are represented simply by their basement membrane, their cells having entirely disappeared. Some pathologists consider that the changes in the tubules of the kidney are dependent on the interference of the circulation through the glomeruli, as the lesions present in the glomeruli in the granular kidney are such as to materially interfere with, and in many cases to arrest, the circulation through the tuft, and the degenerative and necrotic changes observed in the tubular epithelium may very well be looked upon as secondary to the loss of their blood supply. Owing to the nature of the circulation in the kidney, it is obvious that glomerular lesions must interfere to a very great extent with the nutrition of the tubules which are supplied by the efferent vessel leaving the glomerulus.

The arteries in the kidney show extensive lesions, the outer coat is thickened, and there may be a considerable amount of periarteritis. This is especially marked in the long vessels which pass up through the medulla. The middle coat is also thickened, and the thickening, even in the middle coat, is largely dependent on the formation of connective tissue. The greatest thickening, however, of the wall of the vessel is in the sub-endothelial layer, which may increase in thickness to such an extent as to equal that of the other coats of the artery. The fibrous thickening of the sub-endothelial layer is often not uniform, but may be patchy and more marked in one-half the circumference of the vessel than in the other. Thrombosis may occur as a complication in the smaller interlobular arteries, and it may also occur in the renal vein.

The vessels of the body at large present a great variety of lesions, developed to varying extents in different cases. In the form of the disease associated with arterial degeneration, the large vessels, such as the aorta, commonly lose their elasticity, and they may present extensive atheromatous changes. These, however, are to be looked upon rather as the result of arterial disease than as primarily dependent on the kidney lesion, and cases of extreme granular kidney are not uncommonly seen where the general arterial lesions are not well developed. The smaller arteries show signs similar to those described in the renal arteries. In many cases the cerebral vessels present the minute aneurysms that are such a frequent cause of cerebral hæmorrhage. In other cases the cerebral vessels, notwithstanding the general arterial degeneration, are fairly healthy; and the condition of the cerebral vessels cannot be determined with any degree of accuracy from the state of the vessels in the body at large.

The heart is usually hypertrophied, although the degree to which this is present varies greatly in different cases. This probably depends on the degree to which the vessels of the body at large are degenerated. In some cases, especially of primary granular kidney, the hypertrophy of the heart is but slight, notwithstanding the extreme diminution in the size of the kidney. In others with extensive arterial degeneration the cardiac hypertrophy may be very extensive, although the kidneys are not greatly diminished in size; and it would seem as if the degree of cardiac hypertrophy is to be correlated rather with the condition of the arteries of the body at large than entirely with the state of the kidney vessels.

The hypertrophy affects more especially the left side of the heart, but whenever it is extreme in amount the right side is also involved to some extent. The hypertrophy is usually sufficiently obvious to be noticeable to the eye, and in some cases the increase in the size of the heart may be such as to rival the appearances seen in the so-called bovine heart. The hypertrophy is dependent on an overgrowth of the muscular tissue, especially of the left ventricle; but in the later stages of the affection dilatation is apt to ensue, as a result of degenerative changes in the muscular substance. The dilatation is often sufficiently marked to cause incompetence of the mitral valves. Mitral regurgitation, as a result of dilatation of a hypertrophied heart from granular kidney, is not at all uncommon clinically. The most common forms of degeneration are those in which the muscular fibres are directly affected by hyaline and pigmentary changes. In some cases, however, there is an overgrowth of fibrous tissue in the wall of the heart, and in a few cases this may take the form called by the French disseminated sclerosis of the heart, owing to the unequal development of the fibrous tissue in different portions of the heart wall. Both forms of fibroid heart, namely, where the overgrowth is general and diffuse, and, secondly, where it is scattered, are rare.

Symptoms.—The symptoms presented by cases of granular kidney are very various. In a certain number of cases no symptoms of ill health are produced until a sudden complication supervenes, such as cerebral hæmorrhage. In others the first symptoms of illness are uræmic phenomena, developed often with great suddenness; but more usually a patient suffering from granular kidney comes under observation, either owing to impairment of general nutrition, or else owing to disturbance of the circulation. In the type of case presenting signs of nutritional disturbance, the most characteristic symptoms are failure of health, strength, and energy, combined with considerable loss of flesh, without any obvious symptoms pointing to disturbance of the renal functions. Inquiry, however, elicits that micturition is rather more frequent, and that the patient passes considerable quantities of dilute limpid urine. The loss of flesh is often considerable, and may rival in amount that seen in some forms of malignant disease. In other cases the loss of flesh is less marked, and the patient's main symptoms are referred to the digestive system. He complains of loss of appetite, especially in the morning, together with occasional nausea and vomiting; diarrhœa is also not infrequent, and may be very severe. The urine in cases of granular kidney, uncomplicated by mitral regurgitation, is abundant, from 60 to 120 oz.; more usually approximately 80 oz. are passed in the twenty-four hours. Micturition is more frequent than normal, especially at night. The urine is clear, pale, and of low specific gravity, the latter being usually under 1010, and frequently as low as 1005. The specific gravity of the whole twenty-four hours' urine must be, however, observed if any accurate deductions are to be drawn. Crystals of uric acid are not uncommonly deposited. Albumin is present in small quantity, usually not amounting to more than 1 per cent. The quantity of albumin, however, is variable from time to time, and it is clearly established that it may even temporarily disappear. The proteid matter in the urine may not only be in the form of albumin and globulin, which are coagulated by heat, but albumoses are also not uncommonly present, and exceptionally cases are seen where considerable quantities of albumoses are present. In the albumosuria of granular kidney, however, the more usual condition is for traces of these substances only to be present. The cause of the albumosuria in this malady is obscure, but it is possible that it is associated with deranged digestion and absorption from the intestine, so that the albumoses formed during digestion pass into the circulation, and are excreted by the kidney. In some cases the albumosuria in granular kidney is dependent on the coexistence of some other morbid process, such as the presence of an effusion in some part of the body, or, in rare cases, to the presence of bone disease, ovarian cysts, etc. When albumin is absent from the urine, in cases of granular kidney, it is usually the morning urine that is free. Some cases of granular kidney, terminating in death, have been described, where examination of the urine on more than one occasion during the fatal illness has failed to reveal the presence of albumin. Granular and hyaline casts in small numbers are usually present. In some cases of granular kidney, the urine contains considerable quantities of blood; this is rare, but when it occurs it may be sufficiently marked to lead to an erroneous diagnosis, such as malignant disease of the kidney, the presence of the general wasting, together with blood in the urine in comparatively large amount, having led to this error. Hæmaturia, in these cases of granular kidney, may sometimes be dependent on the supervention of acute nephritis; but it is probable that occasionally it is due simply to hæmorrhage from the mucous membrane of the pelvis of the kidney. Such hæmorrhages may be not only very abundant, but may also be sudden in their onset, and transitory in their duration.

In cases of granular kidney coming under observation, owing to disturbance of the nutrition, other signs of the disease are usually well marked, such as a high-tension pulse, and the presence of retinal changes, and more especially hæmorrhages in the fundus. Such patients commonly suffer also from other symptoms dependent on the existence of high tension, and amongst others from throbbing headache, which is greatly aggravated by sudden changes in position.

In the variety presenting cardiac symptoms, the patient usually comes under observation owing to the onset of cardiac irregularity or of dropsy of the cardiac type; and, speaking broadly, such cases have the aspect of mitral disease. It is not an uncommon mistake clinically for mitral disease depending on secondary degeneration of the heart, associated with granular kidney, to be erroneously diagnosed as mitral disease dependent on a primary affection of the mitral valves. This mistake is due to the fact that, where cardiac failure has supervened in granular kidney, some of the most characteristic features of the malady are lost or obscured. Thus the quantity of urine owing to the presence of the dropsy may not be greatly increased, and the tension of the pulse may not be very high owing to the onset of the cardiac failure. Such patients present dropsy of the cardiac type most marked in the dependent parts of the body, and the heart beats irregularly both in force and in rhythm. Cardiac pain, palpitation, and shortness of breath are always prominent.

In cases in which the symptoms of circulatory disturbance are marked, but where the cardiac failure has not progressed to a sufficient extent to cause cardiac dilatation and cardiac dropsy, the patient presents the physical signs of cardiac hypertrophy. The apex-beat is displaced downwards and outwards; the first sound is reduplicated, or preceded by an apparent presystolic murmur, so that a galloping rhythm is developed. The second sound at the aortic cartilage is markedly accentuated, and the pulse is hard, incompressible, and persistent. Extreme high tension may be present without the vessels having undergone any very considerable thickening. In the latter stages, however, where thickening of the vessels is well marked, the vessel wall can be felt like a piece of whip-cord under the finger, when pressure has been exerted on an artery such as the radial; and it is this high-tension pulse, with or without the signs of arterial thickening, that is one of the most important means of diagnosing granular kidney. The examination of the pulse is often of more importance than that of the heart, owing to the fact that extreme cardiac hypertrophy may be overlooked by the coexistence of emphysema, and emphysema often extreme in amount, is a common accompaniment of granular kidney.

When cardiac hypertrophy has become replaced by dilatation, the physical signs are those of a mitral lesion, although usually in such cases a galloping or cantering rhythm may still be well marked. In some of these cases of cardiac hypertrophy, due to granular kidney, the physical signs may present a similarity to those of mitral stenosis, and it is possible that, owing to the enlargement of the ventricle, a condition of virtual mitral stenosis may exist during life. The bases of the papillary muscles are carried away from the mitral orifice, owing to the increase in the size of the ventricle; and in this way the mitral valve may be prevented from opening freely, and thus a condition of virtual mitral stenosis may be brought about. Cases are not uncommonly seen of granular kidney presenting cardiac symptoms, such as palpitation and shortness of breath with no dropsy, and where, together with the physical signs of a presystolic murmur, there are also signs of cardiac hypertrophy. Where dropsy is present, it is a late effect, and resembles very closely that seen in mitral disease, beginning in the legs, gradually spreading up and involving the serous cavities.

Epistaxis, often considerable in amount and occurring repeatedly, is not uncommon in granular kidney, and a smart hæmorrhage will often relieve some of the effects of high tension and more especially the headache. Purpura is also not uncommon, and is especially well marked in the legs. Pigmentation of the skin, like that seen in chronic Bright's disease, is also not very rare.

The cerebral hæmorrhage of granular kidney is generally of a severe type; it may occur, however, not only in the white substance, but also in the meninges. Many cases of granular kidney terminate with uræmic symptoms, and sometimes acute uræmia is the first indication of the existence of this grave malady. Cramp and twitching are also frequent initial symptoms of uræmia. Inflammatory complications, such as pleurisy, pneumonia, and pericarditis are not uncommon. The dyspnœa that is so often seen in cases of granular kidney is sometimes associated with the presence of hydrothorax, but more often it is either of uræmic or of cardiac origin, and when of the former it may very closely simulate asthma, occurring especially at night. Retinal complications, such as retinal hæmorrhages, neuritis, and retinitis, are common. In some instances of granular kidney the existence of this disease is detected owing to the occurrence of sudden retinal hæmorrhage interfering with vision.

Diagnosis.—The diagnosis of granular kidney is often extremely difficult, and not uncommonly the existence of the malady is revealed for the first time in the post-mortem room, as for instance in cases of cerebral hæmorrhage, and in a few cases of uræmia of sudden onset. In the majority of cases, however, the disease has produced symptoms for some time before the fatal seizure. The early symptoms are often so trivial that the lesion may be entirely overlooked, and the patient's troubles thought to be due to neurasthenia. This is especially liable to occur where the disease has occurred in a person who leads a life of strain and worry, and where the prominent symptoms are loss of energy and flesh. In other cases it may be difficult to determine whether the albuminuria is not of the so-called physiological variety. In others presenting cardiac symptoms, the whole trouble may be put down to the heart, when the heart is really affected secondarily through a renal lesion. Diagnosis in all cases should be based on an examination of the urine, controlled by an examination of the heart and arteries, and an examination of the optic fundus. It is important to examine the urine on several occasions, and not to be contented with the absence of albumin on a single examination, before deciding to negative the existence of this disease. High tension is a most important diagnostic symptom.

Prognosis.—Granular kidney is an incurable affection, and although patients may often live for many years after presenting the signs of obvious high tension, and be able to actively engage in occupations which require great vigour of mind, yet such patients may die suddenly at any moment from cerebral hæmorrhage, so that the prognosis in these cases is most uncertain, even when there are no marked symptoms of ill health except the presence of the albuminuria, together with the signs of high tension. Where uræmic symptoms are present, or where the heart shows signs of failing, owing to the development of dilatation, the prognosis is necessarily most grave.

Treatment.—In the treatment of granular kidney, the main indications are to place the patient in such circumstances as to diminish the work of the kidney as much as possible, and to avoid as far as may be the evil effects which result from the high tension. It is very essential in this condition that strains of all kinds should be avoided, owing to the great risk of cerebral hæmorrhage. Further strain should be avoided, owing to the danger of cardiac dilatation supervening; but the two great dangers in this disease are cerebral hæmorrhage on the one hand, and uræmia on the other. In many cases the patients are men of active habits, often living a life of great stress and anxiety, and frequently, in the early stages at any rate, their physical condition is such that they are capable of indulging in more or less severe exercises. It is essential that, as soon as the condition of granular kidney is recognised, such patients should lead an exceedingly quiet life, as free as possible from worries and anxieties, and they should not take part in any severe exercise. Although severe exercise is contra-indicated, owing to the state of the circulation, such patients should be out as much as possible, so as to improve their general health. The activity of the skin should be promoted by baths, and it is very essential that the bowels should be kept freely open, and that all constipation should be avoided; but in the prescribing of purgatives it is essential that no purgative which causes straining or griping should be ordered, as straining at stool is not uncommonly a direct exciting cause of cerebral hæmorrhage. For the relief of constipation, therefore, some gentle aperient or saline purgative should be ordered as a routine measure. It is probably beneficial for these patients to drink fairly freely of simple mineral waters, or water, for the purpose of flushing the kidneys. Alcohol should either be entirely prohibited or only given in very small quantities. In most cases it is better to prohibit it entirely; but where the appetite is poor, and digestion impaired, it may be necessary for small quantities to be given to aid digestion. Tea, coffee, and cocoa may be taken moderately, but all these substances are really diuretic, and it is always a question as to what degree it is allowable to give stimulating diuretics in such conditions. The diet should be light, and the patient should be warned against eating excessive quantities of meat, and this is more especially necessary as many patients suffering from this condition are large meat eaters. It is especially necessary that such patients should not eat highly seasoned and tainted articles of diet, such as high game. Articles of diet, which in an ordinary healthy person may simply cause an attack of diarrhœa or vomiting, may, in a person suffering from renal disease such as granular kidney, cause very serious illness, and, it is said, even death with symptoms of uræmic poisoning. Residence in a warm, equable climate during the winter months is desirable in this affection, as well as in chronic Bright's disease. With a carefully regulated diet, avoidance of anxiety and worry, and residence in a warm, equable climate during the winter months, life may be prolonged for many years.

For the relief of the high tension many remedies have been recommended. As a routine measure, saline purgation is probably the most effective means. The nitrites may be prescribed for the relief of this condition, and nitroglycerin is usually of more avail than nitrate of amyl, owing to the fact that its action is more persistent. More recently, erythrol-tetranitrate has been recommended and used with success, on the ground that its action, though slighter in amount than that produced by nitroglycerin, is more persistent. Although these drugs are capable of lowering the blood pressure, by causing vascular dilatation, yet in this disease, owing to the anatomical changes that have occurred in the arteries, their use is often not followed by the striking results that are seen, for instance, in cases of angina pectoris. At the same time, good effects may be produced, and very commonly the headache, which is a distressing feature of the high tension produced in this disease, is often markedly relieved by this substance. Aconite in small doses may also be ordered, with a view to lowering the tension, by the action of the drug on the heart. It is a drug, however, that has to be used with caution. Iodide of potassium is probably useless as a means of lowering tension.

Cases presenting symptoms of cardiac dilatation are very difficult to treat, since, although they present the general features of mitral disease, the practitioner is on the horns of a dilemma with regard to the use of digitalis or other cardiac tonics in this condition. The heart is failing very largely, as a result of the increased strain to which it has been subjected, and the vessels are often in a brittle condition; and therefore it has been urged that the use of such remedies as digitalis, strophanthus, and strychnine, which raise the general arterial pressure, are most unsuitable, inasmuch as they tend to increase the strain which is already causing the heart to fail. In other words, they tend to increase the work which the failing heart has to do. Digitalis, however, not only contracts the arteries and increases the work of the heart, but, owing to its action in increasing the tonic contraction of the heart, and in increasing the force of the heart's beat, and in slowing the rhythm, it undoubtedly enables the heart to meet the increased strain in a better manner. In cases of granular kidney, in which cardiac dilatation and cardiac dropsy have supervened, digitalis, although from some points of view, and on theoretical considerations, would seem unsuitable, is yet practically a substance which gives the greatest relief. Its administration in these cases, however, should be carefully watched. There is, however, no other cardiac tonic that will take its place, although, on theoretical grounds, strophanthus has been urged by some as a better and more suitable remedy than digitalis. The beneficial effect of digitalis in these cases is probably largely dependent on its action in increasing the tonus of the ventricular muscle, and enabling the heart to expel the blood which tends to stagnate in it. Strychnine is also of value in these cases.

In some cases great restlessness and dyspnœa are present, which, although sometimes of uræmic may be of cardiac origin. Whether of uræmic origin or of cardiac origin, they are most efficiently treated by the administration of morphine or opium in small doses. This drug is one, however, which must be given with extreme caution in this condition, as in all other affections of the kidney; but at the same time the mere existence of Bright's disease, or of a granular kidney, is not of itself an absolute bar to the cautious administration of these drugs. The uræmic symptoms must be treated on the ordinary lines of such a complication, such as free purgation, sweating by a hot-air bath, and pilocarpine, venesection, and administration of chloroform or morphine. Venesection may be of use in the treatment of uræmia, and it will also relieve some of the more distressing symptoms produced by high tension, as for instance the intense headache, sleeplessness, and restlessness. It may also be of service for the relief of cardiac dilatation. It is generally only employed for the relief of uræmic symptoms, but many patients with a granular kidney are often greatly relieved by the occurrence of a spontaneous hæmorrhage, such as epistaxis.

The anæmia that is often present should be treated by the administration of iron and arsenic.

If dropsy of a cardiac type should supervene to a marked extent, it may be necessary for incisions to be made, and these require the very greatest precautions in such cases to avoid septic complications. The tissues of patients suffering from granular kidney and Bright's disease have their resistance very greatly lowered, and erysipelatous and other septic complications not uncommonly supervene, and are exceedingly formidable.

AMYLOID DISEASE.

Lardaceous degeneration of the kidney was formerly included as one of the varieties of Bright's disease, owing to the fact that not only was it liable to be mistaken for Bright's disease, but also because the two lesions frequently coexist in the same kidney. In many cases, lardaceous disease is only an accompaniment of chronic Bright's disease, and lardaceous degeneration in its more severe forms rarely affects a kidney without the epithelial structures of the organ showing some lesions characteristic of chronic Bright's disease. Lardaceous disease of the kidney is not so common as it formerly was, owing to the improvements that have occurred in the surgical treatment of tuberculosis, of suppuration, and of bone disease.

Etiology.—The causation of amyloid kidney is the same as that of amyloid disease in other parts, and it rarely, if ever, exists by itself, the disease affecting commonly the intestine, the liver, the spleen, and other glands, such as the thyroid. Prolonged suppuration, syphilis, and bone disease are the common causes. In some bone diseases—as for instance caries of the spine—amyloid disease of the kidney and other parts may exist to a high degree, when there is not only comparatively little suppuration, but where even the bone disease may be very limited. Waxy degeneration of the kidney associated with Bright's disease is not uncommon as a complication of phthisis, and it is sometimes seen in the kidney as an accompaniment of the nephritis produced by lead or by gout.

Morbid anatomy.—The kidney affected with amyloid disease is usually large. Exceptional cases are seen, in which the organ is but little increased in size; and some have been described where the amyloid change has been marked, and the kidneys have been small and granular. In some cases the increase in size is very great, the kidney weighing 10 oz. or even a pound. The surface of the organ, on stripping the capsule, is smooth, pale, and opaque, often somewhat yellowish in colour, and the stellate veins are exceedingly well marked. On section, the cortex is increased in thickness, and has an opaque, glistening, yellowish white appearance, and the glomeruli are often prominent and stand out so as to be easily visible to the naked eye. The pyramids are congested, and their appearance is in great contrast to that of the cortex. The amyloid degeneration affects the vascular structures of the kidney to the greatest extent, and is said to begin in the capillaries of the Malpighian tufts; and the straight vessels of the medulla, the vasa recta, are affected later. In many cases the morbid process is often entirely confined to the vessels; in others, however, the tubules are affected, and the changes in the tubules are said to be most marked in the basement membrane. The epithelial cells lining the convoluted tubules are not so commonly affected, although they may show lesions similar to those found in chronic Bright's disease, and in some cases these cells have undergone granular and fatty degeneration. Waxy disease of the kidney has afforded some most striking instances of the variety of Bright's disease known as the large white kidney, owing to the great resemblance of the two conditions to the naked eye.

The heart, in lardaceous disease of the kidney, is not hypertrophied except in the rare instances where lardaceous granular kidneys are found, and there the heart may be increased in weight.

Symptoms.—Lardaceous disease of the kidney produces a very similar clinical picture to that seen in chronic Bright's disease, and the differential diagnosis between the two conditions very often largely turns on the coexistence of amyloid changes in other organs, such as the liver and spleen, together with the absence of cardiac hypertrophy. The disease is accompanied by great anæmia, and dropsy is often, but not invariably, present. In many cases the dropsy resembles somewhat that seen in cardiac disease, by being more marked in the dependent parts of the body. If dropsy be absent, the urine is very abundant, over 100 oz. being commonly secreted in the twenty-four hours, and cases have been described where as much as 200 have been secreted. The urine is pale, limpid, and of low specific gravity, under 1010; and where the urine is extraordinarily abundant, albumin is usually present in small amount, so that in these cases the characters of the urine resemble those seen in granular kidney. The condition, however, can be readily separated from that of granular kidney, owing to the absence of cardiac hypertrophy and marked arterial changes. In other cases, where the quantity of urine is not so great, the amount of albumin may be much larger, so that often the condition approximates to that described in the contracted variety of Bright's disease, the urine being pale, abundant, and containing large amounts of albumin. Where dropsy is a marked and an increasing feature of the illness, the quantities of urine secreted are necessarily smaller, and the fluid may be highly albuminous. Granular and hyaline casts are abundant, and cases have been described where the casts have presented the amyloid reaction. The amount of urine is much greater than in Bright's disease. The symptoms seen in these cases are often complicated by those dependent on the disease causing the amyloid degeneration, but very commonly waxy degeneration of the kidney causes ultimately the onset of uræmia. The duration of amyloid disease is very uncertain, as it is so largely influenced by its cause, but it may last from a few months to two years.

Diagnosis.—The disease cannot, as a rule, be diagnosed by mere examination of the urine, as the state of the urine may resemble that seen in granular kidney or in chronic Bright's disease. Usually the condition is recognised by the existence on the one hand of the urinary changes, accompanied by evidence of amyloid disease in other organs, and by the absence of cardio-vascular changes. In cases of pyelitis, the existence of severe albuminuria, together with the absence of cardiac hypertrophy and high tension, is very suggestive indeed of the presence of waxy disease as a complication.

The prognosis of amyloid degeneration depends very largely on that of the underlying disease causing it. If this is amenable to treatment, the waxy degeneration of the kidney or of the liver may undergo arrest, if not cure.

Treatment.—The treatment of waxy disease resembles in some respects that of Bright's disease; thus the treatment of the dropsy and of uræmic complications are the same in both conditions, but in the early stages of waxy disease of the kidney the patient requires a far more liberal diet than that ordered in Bright's disease, and this is the main reason why it is essential to make a differential diagnosis between the two conditions, if possible. In the earlier stages of amyloid disease, characterised by polyuria with a trace of albumin, the eliminating functions of the kidney are not seriously interfered with, and inasmuch as the disease causing the waxy degeneration is usually a debilitating one, a nourishing and liberal diet and even stimulants may be required. In many cases, however, and more especially in the later stages, a similar treatment to that used in Bright's disease is necessary, and this is often rendered necessary by the fact that with the waxy degeneration the lesions of Bright's disease so often coexist.

CYSTS OF THE KIDNEY.

Etiology and morbid anatomy.—A considerable number of different varieties of cysts in the kidney are found. In some the cysts are small in size and few in number; in others a single large cyst is present; in others, and more especially in the congenital form of disease, the cysts are present in such numbers as to make the kidney resemble a bunch of grapes in appearance.

Scattered or single cysts, varying in size from a pin's-head to a pea or sometimes a small cherry, are found, more especially in cases of granular kidney, and are usually situated in the cortex, just beneath the capsule of the organ. Although these cysts are most frequently found in cases of granular kidney, they are also seen in the contracted variety of Bright's disease, and to a slighter extent occasionally in kidneys which present no signs of gross disease. The formation of cysts in granular kidney may be much more abundant, and they may occasionally reach such a size that the organ presents on section quite a honeycombed appearance. The scattered cysts which are so commonly seen in the granular kidney are formed by dilatation of the renal tubules, and it is only an exaggerated condition of the dilatation of the renal tubules that is found, not only in granular kidney, but in most varieties of Bright's disease. There is little doubt that these cysts, at any rate, are formed as a result of obstruction of the uriniferous tubules. The contents of these cysts are usually a clear, amber-coloured, slightly albuminous fluid, containing traces of urea. In some cases the contents are dark in colour, from the presence of extravasated and altered blood pigment, and in others the contents are of jelly-like consistence. The fluid in these cysts is often pent up under pressure. These cysts, occurring to a slight extent in healthy kidneys, and to a greater degree in granular kidney and in Bright's disease, are of no great importance, except as demonstrating that such cysts can arise as a result of obstruction to the uriniferous tubules. Although these cysts arise as a result of obstruction, there must be a considerable formation of new epithelium, inasmuch as they are lined by a uniform layer of cubical hyaline nucleated cells.

Single large cysts, sometimes as big as an orange, and possessing fairly thick walls, are occasionally found. Such cysts occupy usually one or other extremity of the kidney, and are perhaps more usually situated towards the anterior surface, so that the bulk of the kidney substance lies behind them. In most cases of single large cysts, the remaining kidney substance is quite healthy; but in some cases the organ is deformed in shape, not only from the presence of the cyst, but also shows signs of some congenital anomaly in form. These large cysts are usually held to be dependent also on obstruction of the uriniferous tubules.

They are of no great importance clinically, except that they may be of sufficient size to form tumours that may be recognised on clinical examination. If present on the right side, they may be mistaken for an enlarged gall bladder. In cases presenting any renal symptoms, such as pain or hæmaturia, perhaps dependent on some other cause, the detection of a tumour really due to a cyst may lead to an erroneous diagnosis, *e.g.* growth. It has been thought by some that these single large cysts of the kidney may sometimes be due to external violence, the kidney undergoing a slight rupture, and the cyst forming at the seat of the injury. The fluid in these cysts does not usually present any close resemblance to urine in its composition, inasmuch as the fluid is albuminous and sometimes gelatinous, and contains only traces of urea and other nitrogenous extractives; it may also contain considerable quantities of cholesterin.

General cystic disease of the kidney, where the kidney tissue is apparently more or less replaced by a mass of cysts of varying size, exists in at least two and probably more forms, one in which the lesion is congenital, and a second in which the condition only becomes obvious in adult life or even in the elderly. Some pathologists consider that the two forms are really identical, and that the adult condition is merely the result of a congenital lesion which has remained latent for a great number of years. The main argument in support of this view, apart from the general similarity of the condition in the two cases, lies in the fact that the adult condition may remain latent throughout, and may be only recognised after death, the patient not necessarily succumbing to the renal condition, but dying accidentally, or from some other disease. In many cases, however, the cystic disease affecting the kidney is itself fatal, and in such cases it is difficult to suppose that the condition has really existed from fœtal life.

In the congenital form of the affection the kidneys are found of all sizes, in some cases being not more than doubled or trebled in size, and presenting on section the appearance of a pomegranate. In others the size of the cystic kidneys is such as to produce an impediment to delivery, and in such cases the tumours are enormous, filling the abdomen and forming one of the well-recognised causes of difficult delivery, necessitating evisceration. In this condition both kidneys are affected, although the increase in size of the two organs may not be equal. Virchow supposed that the so-called cystic degeneration seen in fœtal life was dependent on obstruction of the straight tubules, and subsequent dilatation of all tubules, as a result of the inflammation produced by uric acid crystals. Other observers have concluded that the condition is associated with errors of development, such as persistence of the Wolffian body, and that the cyst formation occurred in the remnants of this. The cysts in all these cases are separate from one another, they are lined with a flattened cubical epithelium, and they contain a fluid which, although it may contain traces of urea, is often highly albuminous, sometimes gelatinous, and in the case of the larger cysts frequently contains altered blood pigment. Malformations of the kidney and of the lower urinary tract, the ureter, bladder, or urethra, are frequently present together with congenital cystic disease of the kidney. In many such cases the renal calyces and pelvis of the kidney are absent or ill developed. Cases have been described with malformations in other parts, such as the presence of an encephalocele, and the presence of supernumerary fingers and toes, and other anomalies.

In the form of the disease seen in adults, the affection is nearly always bilateral, although occasionally cases have been described in which the disease was unilateral. The kidneys are enormously increased in size, so as to measure from above down 12 or 15 in., and they present but little resemblance to a normal kidney, consisting as they do of an agglomeration of cysts, varying in size from a split pea to a large walnut. The contents of the cysts are various, in most cases of an amber colour, in others darker from altered blood pigment, in others quite black. These varieties in the colours of the cyst contents produce a variegated appearance in the colour of the mass. The cysts are entirely distinct and separate from one another, and also from the pelvis of the kidney, so that their contents cannot be emptied by pressure. On close inspection of the section, remains of renal tissue can be seen in between contiguous or adjacent cysts, and, thanks to this, a considerable amount of renal tissue, although in an attenuated form, may exist scattered between the neighbouring cysts in an organ which at first sight presents no resemblance to a kidney. On microscopic examination, the cysts are found to be lined with a flattened epithelium, and the remains of kidney substance lying between adjacent cysts may show signs of fibrosis.

All distinction between the cortex and the medulla is lost in cases of advanced cystic disease; in some, however, it may be detected that the cyst formation is more marked in the cortex than in the medulla; in other and rarer cases the converse is the case, the cystic disease being much more marked in the medulla than in the cortex, and the writer has seen one case where the medulla was extensively affected and the cortex nearly intact.

In some cases of general cystic disease of the kidney, small cysts may be found underneath the mucous membrane of the pelvis and of the upper part of the ureter; and in a certain proportion of cases of cystic disease, cysts are found in other parts of the body, more especially in the liver, the brain, and occasionally in the thymus and other glands. The liver, however, is the organ which is more especially liable to contain cysts, and in some instances they reach quite a high degree of development; and in such cases this organ commonly shows the presence of overgrowth of fibrous tissue. It is a point of some importance in the pathology of this condition, that the cystic disease which advances to such a high degree in the kidney is found in other parts of the body, such as the liver and brain.

The heart in cases of general cystic disease may be hypertrophied, but this is not constant, and the arteries do not show the marked changes associated with the extreme forms of granular kidney and of some varieties of Bright's disease. This is a fact of some importance, as regards the view frequently held, that the cardiac hypertrophy in renal disease is entirely dependent on the diminution of the renal substance. General cystic disease and hydronephrosis afford two examples of morbid processes which greatly diminish the available amount of renal tissue, but in neither case is cardiac hypertrophy a marked or invariable result.

The pathology of these cases is exceedingly obscure. It is thought by some that they arise in the same way as the congenital form, either as a result of obstruction of the straight tubules from atresia, perhaps produced by inflammatory trouble, or else as a result of the cystic degeneration of the embryonic Wolffian bodies. It is possible, however, that cystic disease of the kidney is really more of the nature of a growth, and the presence in some of the typical cases of cysts in other parts of the body, such as the liver, might be used as an argument in favour of this view. Certainly, some cases of cystic disease are dependent on the formation of adenomata in the kidney, which subsequently undergo degeneration, with the formation of cysts, and in some of these cases secondary deposits, with an adenomatous structure, may occur. The main argument against cystic disease being really of the nature of a growth, may be found in the fact that it is usually bilateral, but this is not conclusive, as bilateral disease of the nature of growths may occur in other organs, *e.g.* the ovary.

Symptoms.—The adult form of the disease produces in a great number of cases no symptoms at all, and the condition is discovered accidentally in the post-mortem room. In other cases the disease runs a latent course, and then suddenly uræmic symptoms develop, causing death; and in a third group of cases the condition produces more or less marked symptoms, which in a number of cases may enable the disease to be diagnosed. In the last group of cases, where symptoms are present, they may be present for a considerable number of years, as for instance in one case the condition was diagnosed four years before it caused the death of the patient from uræmia. In the great majority of cases, however, symptoms are not produced until towards the termination of the patient's life, and life is not prolonged for more than a few weeks or months after the first onset of symptoms. The most common effects produced by the disease are, first of all, a general loss of strength and energy, together with pain in the back. Soon nausea and vomiting may develop, and the patient seeks advice, owing to these symptoms. In a small proportion of cases the occurrence of hæmaturia, often profuse, is the first indication of ill health, and in the cases of general failure of strength hæmaturia is not uncommon. The most characteristic symptoms produced by the disease in adults are the general failure of strength, emaciation, pain in the back, and hæmaturia. Dropsy is rare, but occasionally very slight pitting of the skin may be present. The urine is abundant, pale, of low specific gravity, and commonly contains traces of albumin; in other words, it resembles the urine that is secreted in cases of granular kidney. In some cases the first symptom that has attracted notice has been the development of intestinal obstruction, from the pressure of the renal tumours on the colon.

The main interest of the condition, from a clinical point of view, lies in the fact that it is a disease in which the amount of kidney tissue is very greatly diminished, but the remaining portion is not profoundly disorganised. The symptoms the patient suffers from, as the result of this lesion, are loss of strength, emaciation, with the excretion of an abundant dilute urine; and the fatal termination is commonly uræmia. The uræmia in cases of cystic kidney is often extremely sudden in its onset, and, as mentioned above, it may be the first symptom of ill health. In such cases the uræmia is usually of the acute or fulminating type, and characterised by the occurrence of epileptiform seizures, delirium, and coma. In others, especially where the malady has already produced the symptoms described above, the uræmia resembles more the latent uræmia seen in calculous obstruction, and in other diseases of the kidney. The patient then suffers from slight nausea, cramps, twitchings of the muscles, the temperature becomes subnormal, and the pupils contracted; but the grave and severe symptoms which are usually seen in acute uræmia do not necessarily occur in this variety. In most cases, dyspnœa is a marked symptom towards the end; the patient becomes more drowsy and finally comatose, but death may occur suddenly, from respiratory failure, in such cases without coma.

In cases of cystic kidney, where uræmia supervenes, the quantity of urine is usually diminished, but complete suppression may not occur even in fatal cases.

Diagnosis.—In many cases, owing to the absence of symptoms, the condition is not diagnosed. In others, where the malady is latent until it produces intestinal obstruction, errors of diagnosis may arise unless both loins be carefully examined, and a tumour found on both sides, which is one of the most characteristic features of the disease. In the majority of cases the palpation of the abdomen reveals the nature of the tumour, not only owing to its having the characters of bilateral renal tumour, but also owing to the ease with which the irregularities on the surface, dependent on the individual cysts, may be detected.

Prognosis.—The prognosis of the condition depends mainly on whether the malady is causing symptoms. Where symptoms are produced, the disease usually destroys life in the course of a few months, but a case has come under the observation of the writer where the condition was recognised ten years before death. In all cases, however, there is always a risk of the sudden onset of uræmia.

Treatment.—In the rare cases where the disease is unilateral, the kidney may be removed; and such cases have been recorded, where the patient has remained alive and well several years after the operation. Surgical interference, however, even of the nature of an exploratory laparotomy, is dangerous where the kidneys are both affected, and may precipitate an attack of fatal uræmia. The disease is beyond treatment except in so far that it should be treated dietetically and hygienically, in the same manner as cases of Bright's disease, but there is no treatment known to modify or arrest the course of the malady.

HYDRONEPHROSIS.

Hydronephrosis is a condition in which the kidney is distended with pent-up urine, as a result of obstruction, usually of the ureter only, but sometimes of the bladder or urethra as well. Hydronephrosis may be congenital or acquired, but even when acquired it is not uncommonly dependent on some apparently slight congenital malformation.

Etiology.—Congenital hydronephrosis is not uncommonly dependent upon an imperforate urethra, and in this condition the kidneys may undergo such extreme dilatation as to offer a mechanical impediment to birth. In a large proportion of all congenital cases, the hydronephrosis is double. Congenital hydronephrosis, like congenital cystic disease of the kidney, may be associated with malformations in other parts of the body, such as club foot, imperforate anus, etc.

Acquired hydronephrosis is always dependent on an impediment to the exit of urine, and in the great majority of cases the obstruction is situated in some part of the ureter, and even as low down as its entrance into the bladder. The most common cause is obstruction arising from the impaction of a stone, and it is not very rare for a stone to become impacted at the point of entrance of the ureter into the bladder, although it is more usual for the obstruction to be at the point where the ureter leaves the renal pelvis. In other cases, the obstruction is due to the pressure of a tumour on the ureter, and this is more especially the case in uterine carcinoma and fibroids. The cicatricial contraction and thickening of the fibrous tissue of the pelvis, following on attacks of pelvic peritonitis and cellulitis, is also not an uncommon cause. Occasionally, an ovarian tumour has caused obstruction to the flow of urine in the ureter.

Thickening and obstruction of the ureter itself, as a result of inflammation of its walls, dependent on the former passage of a calculus, is found in some cases. The ureter may also become kinked, as a result of adhesion to surrounding structures, following on an attack of peritonitis. In a considerable proportion of cases, some comparatively trivial anomaly is present, such as a supernumerary renal artery, passing over the anterior surface of the ureter, or the ureter is preternaturally narrow, and is implanted at an angle into the wall of the renal pelvis. It is more especially in these conditions of slight anomalies in the arrangement of the renal artery and of the ureter, that the existence of movable kidney acts as a predisposing cause for the production of hydronephrosis.

Some remarkable cases of intermittent hydronephrosis, occurring simultaneously with the menstrual periods, are occasionally seen. In some of these cases a history of a former attack of pelvic peritonitis or cellulitis may be obtained, and it is probable that in such cases the slight swelling of the pelvic organs produced during the menstrual period, together with the adhesions resulting from the former inflammatory disorder, are sufficient to mechanically kink the ureter temporarily.

In cases in which the obstruction of the ureter arises from the presence of tumours in the pelvis, as in carcinoma of the uterus and fibroids, the hydronephrosis is often double.

Morbid anatomy.—Obstruction of the ureter leads first of all to dilatation of the ureter and of the renal pelvis. Soon the papillæ of the pyramids become flattened out and compressed, and this process goes on, until finally the kidney is distended into a sac of very varying size but often as big as a man's head. On section of the dilated organ, it is found to be sacculated, the partitions separating the various loculi being formed by the connective tissue accompanying the blood vessels as they pass up between the pyramids. In advanced cases the kidney may be distended so as to form a mere sac, the wall of which, in places, may not be more than one-eighth of an inch in thickness. This extreme distension of the kidney only supervenes in cases in which the affection is unilateral. Where the cause operates bilaterally, the degree of the distension of the kidney is, except in the congenital cases, more moderate, the medullary portion of the kidney being flattened out but the cortex still remaining distinct, and the enlargement of the organ very much more moderate.

A very considerable atrophy of the tubular structures of the kidney ensues as a result of the obstruction, many tubules shedding their cells, and in others the cells become hyaline or glass-like, losing all their characteristic structure. The glomerular chambers become very considerably dilated, the capillary tuft remaining in the centre of a large dilated chamber. Although very extensive changes take place in the epithelial structures of the kidney, yet a considerable amount of renal tissue remains spread out, forming the wall of the sac. In some cases, especially where the kidney is not greatly enlarged, there is a certain amount of interstitial inflammation, especially in the medullary portion of the kidney between the tubules, and, as a result of this, an overgrowth of fibrous tissue may occur, causing considerable induration and overgrowth of the fibrous tissue of the organ.

When the ureter is obstructed experimentally in animals, as by ligature, hydronephrosis or pyonephrosis invariably supervenes; but in the human subject the impaction of a calculus in the ureter is not always followed by the development of hydronephrosis. Complete suppression may ensue in a certain proportion of cases. It is rather remarkable that, experimentally, obstruction of the ureter, even when complete, fails to produce permanent suppression, although in the human subject such may certainly continue for as long a period as ten days without the supervention of hydronephrosis.

Experimentally, the most characteristic changes produced in the kidney, apart from the mere distension of the organ, are the changes which occur in the cells of the tubules; these undergo atrophy, and in addition they lose their characteristic structure, and become hyaline and glass-like.

If in the hydronephrosis produced experimentally the obstruction is relieved by draining the ureter, a genuine atrophy of the kidney is produced. The kidney returns very completely to its former shape, but is very much reduced in size, and the urine secreted by such an atrophied kidney, produced experimentally, is very small in quantity and low in specific gravity. The fact that such a complete atrophy can be produced experimentally, as a result of ligature of the ureter, with subsequent drainage of the hydronephrosis produced, may perhaps throw some light on the atrophy of the kidney that is not uncommonly seen in the kidney in the human subject as a remote effect of calculous disease.

The fluid that is contained in hydronephrotic kidneys in the human subject is usually limpid and of a low specific gravity, containing traces of urea and other urinary ingredients, and, in addition, it is usually albuminous. In some cases hæmorrhage may occur into the hydronephrosis, so that the fluid contains more or less altered blood pigment, and in rare cases the hæmorrhage is so severe in amount that the whole of the hydronephrotic tumour becomes converted into one large laminated blood clot, producing the appearance of a solid tumour. In cases of unilateral hydronephrosis, the opposite kidney usually shows signs of hypertrophy. In the slighter forms of hydronephrosis, more especially where the disease is double, the distension of the kidney is small in amount, and the fluid contained resembles very dilute urine in its composition.

Symptoms.—The symptoms of hydronephrosis depend very largely on its cause. In cases where slight degrees of the condition are produced by the pressure of pelvic tumours, such as uterine carcinoma, and where the obstruction to the ureters is only partial, the only evidence of the existence of this grave complication is frequently the passage of very dilute urine. Where a complete hydronephrosis is produced on one side, however, the examination of the urine is negative, owing to the fact that the opposite healthy kidney produces the normal quantity of urine, the secretion of the affected kidney being pent up behind the obstruction.

Pain is usually a more or less prominent feature, more especially where the disease supervenes as a result of calculous obstruction, but, in addition to the pain produced by the obstruction, pain in the back and severe pain in the hypochondrium are often present, more especially where the kidney undergoes great distension.

Nausea and vomiting are often also prominent symptoms, and these may be so severe as to suggest the presence of intestinal obstruction. In some cases constipation is produced by the pressure of the renal tumour on adjoining viscera, and, in at least one case, hæmatemesis is said to have occurred as a result of the pressure of the tumour on the vessels of the stomach. In the majority of cases the presence of a renal tumour can be detected, and hydronephrotic tumours are characterised, not only by conforming to the usual type of a renal tumour, but also, owing to their very rounded and smooth outline. Of all the varieties of renal tumours, they are the ones which are most likely to fill up the loin, so that on palpating the abdomen with one hand on the loin, and the other on the anterior surface of the tumour, the whole mass can be easily felt, and fluctuation often detected. In some cases, more especially in movable kidney, or in cases where the hydronephrosis depends on obstruction caused by some pelvic inflammation, the hydronephrosis is intermittent, and the patient gives the characteristic history that suddenly a large quantity of urine is passed synchronously with the subsidence of the tumour.

As mentioned above, these cases of intermittent hydronephrosis sometimes recur synchronously with the menstrual periods; more often it is difficult to account for the intermittence, but when this symptom is present it is very characteristic.

The amount of pain that is felt in cases of hydronephrosis depends mainly on two facts—first, whether the hydronephrosis is produced rapidly; and, secondly, whether the distension of the kidney reaches a very high degree. If the two conditions are combined, the amount of pain may be very great, in others it is not a prominent symptom.

Fever and rigors are not usually present, except as a result of septic infection, and the conversion of the hydronephrosis into a pyonephrosis.

Where the condition is double, in adults uræmia supervenes and is usually of a chronic type, the patient becomes delirious, and suffers from twitchings and other convulsive seizures, the temperature becomes subnormal; coma and death ultimately supervene. This is not an uncommon mode of termination in cases of carcinoma of the uterus, where the ureters have been involved.

Diagnosis.—In cases of intermittent hydronephrosis, the occasional passage of a large quantity of urine, together with the temporary subsidence of the tumour, renders the diagnosis easy, as there is no other condition with which it may be confounded.

In other cases, where the condition is persistent, the diagnosis is often exceedingly difficult, and the tumour may be mistaken for tumour of another organ, such as the spleen, or liver, or for an ovarian tumour; or the nature of the tumour may be mistaken, so that it is looked upon as a pyonephrosis, or as due to a solid enlargement of the kidney. Where the hydronephrotic tumour is of very great size, it may be confounded with localised ascites, but the most common mistake is for the hydronephrosis to be erroneously diagnosed as a splenic, ovarian, or hydatid tumour.

It is liable to be confounded with splenic tumour, owing to the fact that occasionally splenic tumours are vertical in position. The renal origin of the tumour can generally be recognised, if attention be paid to the points, that renal tumours, and more especially hydronephrotic tumours, possess rounded margins, that they are slightly movable on respiration, and that the colon can often be demonstrated by palpation or percussion to pass over the front of the tumour. This is, however, not an absolute criterion between splenic and renal tumours, as occasionally the colon may pass over the tip of an enlarged spleen. The fact that the tumour fills up the loin, and the fact that it is possible to pass the hand below it, are valuable means of distinguishing it from ovarian tumours. A hydronephrotic tumour may sometimes be confounded with a perinephritic abscess, but in the latter condition the pain, the fever, and the constitutional disturbance are usually greater, and the mass is generally more fixed.

Prognosis.—The prognosis depends mainly on whether the affection is unilateral or bilateral; in the former condition the disease is not necessarily dangerous to life, but the cause that has produced the unilateral hydronephrosis may be one that may operate bilaterally, as in the case of calculous disease, and this risk must be borne in mind. Where both kidneys are affected, as in carcinoma of the uterus, the prognosis is necessarily very grave.

Unilateral hydronephrosis may be fatal from the effects of the pressure exerted on surrounding organs, *e.g.* intestinal obstruction, more commonly perhaps by the fluid becoming infected and so becoming converted into a pyonephrosis. Rupture of the sac is exceedingly rare, but has occurred.

Treatment.—The treatment of hydronephrosis is usually surgical, and consists either in tapping the distended kidney, or in draining it, or in the performance of nephrectomy. Draining the kidney is the procedure that is usually adopted, and, during the course of the operation, search should be made for stone, as the impaction of one is such a frequent cause of this condition. In many cases, however, the kidney is so disorganised, as a result of the obstruction, that it is of little service to the patient, and, provided the other kidney is sound and performing its functions properly, it may be desirable to remove the hydronephrotic kidney. The more usual procedure is to drain the kidney first, and then, if the obstruction cannot be found, or if, as a result of the operation, the obstruction is not relieved, the remains of the kidney may be removed at a second operation, although considerable difficulty may be experienced as the result of adhesions to surrounding organs; and in some cases, both of hydronephrosis and of pyonephrosis, it may be desirable to remove the kidney without prior drainage.

NEW FORMATIONS.

Etiology and morbid anatomy.—The kidney is occasionally the seat of either simple or malignant growths; the former are merely of pathological interest, and are of no very great clinical importance. Adenomata, fibromata, and gummata are by no means uncommon, and adenomata, in the form of small growths, from the size of a pin's head to a split pea, are not uncommon in cases of granular contracted kidney, and even in healthy kidneys; they occasionally reach a larger size. They are mainly of interest from the fact that some of the more malignant forms of renal growth have also an adenomatous structure.

Fibromata are seen in the form of small fibrous growths in the pyramidal portion of the kidney, and also in the rare form of multiple growths in the capsule of the organ. The latter are sometimes associated with fibromata of the ovary and of other parts.

Small lipomata are also occasionally seen, but some growths described as lipomata are really instances of tumours occurring in adrenal rests, the cells of which are loaded with fat. The removal of the fat in these cases will reveal the typical structure of the adrenal growths.

Angeiomata are occasionally found. Leukæmic tumours and lymphadenomatous secondary growths are also frequently present in cases of leukæmia and of Hodgkin's disease.

All these growths are mainly of pathological interest. The more serious growths affecting the kidney are adenoma, sarcoma, and carcinoma. Sarcoma may occur *in utero*, the child being born with a large sarcomatous tumour occupying the region of one kidney. They are also not uncommon in childhood, but they may occur in the adult.

True cancer of the kidney is most frequently seen after 40 years of age. Carcinoma of the kidney is more common in males than in females, but there is no great difference in the frequency of affection of either kidney; according to some writers, the right kidney is affected a little more frequently, according to others the left.

The sarcomata affecting the kidney are sometimes the rare form known as alveolar sarcoma, more commonly they are round or spindle-celled sarcoma. Not infrequently the tumour mass contains fibres of striated muscular tissue, and to these tumours the name of rhabdomyoma has been given. This form is not uncommon in the congenital variety of the disease, but it is also found in the sarcomata affecting the young. Many of the congenital renal tumours contain such varied elements that they are often better described as adenomata than as sarcomata.

Melanotic sarcoma, in the form of secondary nodules affecting this structure, when the primary seat of the disease is in some other part, as for instance the eyeball, is occasionally seen. Melanotic sarcoma affecting the kidney is in some cases accompanied by melanuria, but melanuria is not characteristic of the condition, and it may occur apart from the presence of melanotic sarcoma in the kidney, the morbid growth being in some other organ, as for instance in the liver; and melanuria is said to have occurred without the presence of melanotic sarcoma in any part of the body.

The carcinomata affecting the kidney present considerable differences in microscopic character: in some cases they are of the ordinary encephaloid variety, rich in cells, the amount of fibrous tissue separating the groups of cells being small in amount. In other cases the growth has more of the characters of a malignant adenoma, the cells being arranged in such a manner as to simulate a glandular structure.

In most of the cases of carcinoma of the encephaloid variety, the growth is exceedingly soft, and in places is apt to undergo colloid degeneration; in some instances necrotic changes occur leading to the breaking down of the growth, with the formation of abscess-like cavities; in others, hæmorrhage has occurred,—with the result that the section of cancer of the kidney presents a great variety of appearances. In rare instances the formation of fibrous tissue occurs to such an amount as to entitle the growth to be described as a scirrhus.

Epithelioma is occasionally met with, and here the cells are usually of the cylindrical or columnar form, enclosing a lumen, so that the morbid growth simulates somewhat closely the appearance of a tubular gland. Squamous epithelioma, affecting the kidney primarily, has also been described. In one case, recorded by Morris, the morbid growth began in the pelvis of the kidney, and the secondary deposits, which were widespread in the body, repeated the structure of a squamous epithelioma.

Malignant growths which arise in accessory suprarenal bodies, so-called adrenal rests, resemble sarcomata and adenomata in appearance. These adrenal rests are not uncommonly situated in the cortex of the kidney just beneath the capsule, and growths affecting them, even when of large size, may sometimes be dissected away from the kidney substance; in other cases they grow into and infiltrate it. These growths always contain a considerable amount of fat, but when this has been removed they show the typical structure of the suprarenal body; and this structure is also found in secondary growths, which may be found in the glands and in other parts.

Villous tumours of the renal pelvis are sometimes found, and in some cases the growth of these may be so luxuriant as to form a large mass, which spreads up into the calyces of the kidney, and may in this way produce a very large renal tumour.

Myxoma of the kidney has also been described.

Malignant disease of the kidney usually begins in the cortex of the organ, and more frequently at one extremity. The different forms spread with varying degrees of rapidity, but very often the shape of the organ is maintained, notwithstanding the large size the growth reaches. In other cases the growth spreads through the capsule of the organ, and spreads by mere continuity to the adjacent retroperitoneal structures, but both sarcomata and carcinoma may, however, reach a very large size without the disease spreading beyond the capsule of the organ. In some cases of epithelioma and of papilloma, the disease, however, begins in the renal pelvis, and spreads into the substance of the organ through the calyces. The growths beginning in the accessory suprarenals do not generally run a rapid course, and they frequently enlarge in such a manner as to push the kidney substance before them, so that they can be removed. In others the growth is large, and it is probable that some cases described as renal sarcomata are really of adrenal origin.

Malignant disease, especially carcinoma, has a great tendency to spread into the renal vein, and through this into the vena cava; and the growth may in this way not only block the renal vein on the affected side, but also its fellow on the opposite side, together with the whole vena cava. In a considerable proportion of cases, estimated by most authorities at a little less than one-half, the growth remains limited to the kidney. In others, secondary deposits have occurred, and the infection, as in other cases of cancer, occurs through the blood vessels or through the lymphatic system. Owing to the frequency of the involvement of the renal vein by the growth, the vascular system often plays a great part in the dissemination of the secondary growths. Secondary deposits are found most often in the lungs, then in the liver, and then in the lumbar glands.

Symptoms.—The benign tumours of the kidney usually give rise to no symptoms. In some cases of adenoma, affecting the organ extensively and undergoing cystic degeneration, the cases may present clinically the symptoms of Bright's disease, or those of so-called cystic degeneration of the kidney; such cases, however, are very rare. In cases of papilloma, or villous tumour of the pelvis of the kidney, the only symptoms produced are hæmaturia and attacks of pain, simulating those of renal colic, and dependent on the detachment of portions of the tumour mass and their temporary impaction in the ureter.

In malignant disease of the kidney the characteristic features are pain, together with hæmaturia and the presence of a tumour. In some cases a varicocele has also developed, dependent in all probability on the obstruction to the return of blood from the spermatic vein. Varicocele is seen, not only when the renal growth affects the left kidney, but also when the disease is on the right side, and in both cases is dependent on the tumour mass interfering with the return of the blood through the spermatic vein; although, when the disease is on the left side, this explanation is more simple, owing to the fact that the left spermatic vein opens into the renal vein. In the great majority of cases the renal tumour can be detected at the time the patient comes under observation, and often it is of very large size. It presents the usual characteristics of a renal tumour, having a rounded outline and filling the loin, the latter characteristic being most evident on bimanual examination. The colon can frequently be detected by palpation in front of the tumour mass. The tumour moves slightly with respiration, and this occurs, although to a less extent, even when it is adherent to the parietes. Although such renal tumours move with respiration, they are not nearly so movable as hepatic or splenic tumours. The tumour is sometimes of sufficient size to be seen through the abdominal parietes, on inspection. The position of the intestine, when it is not possible to make it out by palpation, can sometimes be determined by percussion.

Pain is commonly present, but not universally, in cases of malignant disease. The pain is usually of the lumbar variety, although sometimes attacks, simulating those of renal colic, may be present, and are usually dependent on hæmorrhage or on portions of a friable growth being detached. Hæmaturia occurs in at least half the cases, and it is often very profuse in amount and frequently repeated. Sometimes, however, it is very scanty, and it may be absent. It is rather characteristic of the disease for attacks of hæmaturia to occur from time to time, lasting several days.

In rare cases, fragments of the growth can be detected on microscopic examination of the urine; more frequently the urine presents no signs of disease other than the presence of blood. In some cases, owing to thrombosis involving the renal veins, suppression of urine may occur.

Malignant disease of the kidney is of very variable duration, usually under one year; carcinoma of the organ affecting adults may last for several years, and notably longer than in the case of cancer of the liver, stomach, or uterus. In sarcoma affecting the young, the course of the disease is usually very rapid. Death may occur as a result of the asthenia and cachexia produced by the disease; in other cases, from the weakness caused by the repeated hæmorrhages; intestinal complications, such as obstruction, sometimes supervene, from the mass involving and occluding some portion of the intestinal tract; in rare cases the disease spreads, and by involving the vertebral column, produces paraplegia.

In cases where malignant disease of the kidney has spread through the renal vein into the vena cava, thrombosis of the latter may occur, producing all the usual effects which follow in this condition, such as œdema of the legs and distension of the veins in the anterior abdominal wall. In some cases of malignant disease of the kidney, ascites occurs. This may be dependent either on pressure on the portal vein, or on the supervention of peritonitis, from the occurrence of secondary deposits in the peritoneum.

Diagnosis.—The diagnosis is generally easy, if the patient presents a combination of the presence of a renal tumour with repeated hæmaturia and cachexia. Even in such cases, however, mistakes will occur, as in one observed by the writer, where hæmorrhage occurred into a hydronephrotic kidney, and where lamination of the blood clot converted the sac-like kidney into a solid tumour. In this instance the hæmorrhage was dependent on the presence of a stone, and the patient presented a large rénal tumour with hæmaturia. The main difficulties in diagnosis occur where a tumour is present but there is no renal hæmorrhage, or where renal hæmorrhage occurs and no tumour can be detected. In the latter case the diagnosis is often impossible, and the disease may be confounded with calculus, or even with the granular kidney, since copious hæmorrhage may sometimes occur in this malady; and the writer has seen this mistake made.

In cases where a tumour is present without hæmaturia, the diagnosis resolves itself into the differential diagnosis of renal tumours from those of the liver, spleen, and other organs, such as the ovary. Renal tumours may readily be distinguished from ordinary splenic tumours, but in some cases of splenic enlargement the spleen becomes vertical instead of oblique, and in such cases the resemblance between an enlarged spleen and a renal tumour is marked. Renal tumours as a rule possess either a rounded edge, or else the shape of the organ is maintained; splenic tumours usually present a sharp edge.

Splenic tumours are generally more movable with respiration than renal tumours, and the colon is much more frequently in front of a renal tumour than in front of a splenic tumour, although occasionally this portion of the gut may lie over the front of a splenic tumour. The dulness of a renal tumour posteriorly passes right up to the middle line, that of the splenic tumour does not. The angle which a renal tumour makes with the costal margin in front is not so acute as that formed by a splenic tumour.

By attention to these points a differential diagnosis can usually be made. In cases where the mobility of a renal tumour with the respiratory movements is very slight or absent, it may be almost impossible to distinguish between a renal tumour and a retroperitoneal growth; the latter, however, is usually of more irregular outline, and may encroach more on the middle line.

Treatment.—The treatment of malignant disease of the kidney rests with the surgeon, and the results of removal are on the whole unfavourable, probably owing to the fact that by the time the condition can be diagnosed the mass has reached a large size. In some cases, however, more especially where the growth has occurred in an adrenal rest, the tumour has been removed with success. Inasmuch as the only chance of surgical treatment is to remove the kidney early, it is proper that an exploratory incision should be made in all doubtful cases, to determine the nature of any enlargement which may be detected. In some cases where malignant disease has been diagnosed clinically, it has been found that the supposed tumour was really dependent on distension, induration, and suppuration of the kidney, produced by calculous disease, and that it could be successfully removed.

The statistics for the removal of sarcoma of the kidney are a little more favourable than those for carcinoma, but recurrence is prone to occur rapidly.

RENAL TUBERCULOSIS.

Etiology and morbid anatomy.—Renal tuberculosis may exist in several forms—First, the kidney may contain a considerable number of scattered miliary tubercles in cases of general tuberculosis. Secondly, the kidney may be affected along with other portions of the genito-urinary tract, more especially the testicle, the bladder, the vesiculæ seminales, and the prostate. Thirdly, the disease may be limited to the kidneys, or even to one kidney.

In the first group of cases the infection of the organ is through the blood, and the scattered tubercles may be found more or less uniformly throughout the kidney, but more generally in the cortex, just beneath the capsule, only a small number being found in the medulla. They are usually small and whitish yellow in colour; in some cases they may reach the size of a split pea, but it is rare, where the mischief is part and parcel of a general tuberculosis, for them to reach a larger size or to have undergone caseation sufficient in amount to destroy any appreciable amount of the kidney substance.

The great majority of cases of tuberculous disease of the kidney consists of those in which the lesion is one affecting either the kidney only, or the kidney together with some part of the genito-urinary tract, and more especially the bladder, prostate, and testes. The lesions presented by the kidney in the latter cases are diverse, but most frequently a tuberculous pyelitis is present, scattered tubercles being found beneath the mucous membrane of the pelvis of the kidney, and in many cases the disease spreads into the papilla and the adjacent portion of medullary substance, so that masses of caseating tubercle, varying in size, are found in this situation. In still more advanced cases the tuberculous disease in the pelvis of the kidney or in the ureter has led to obstruction of the ureter, and the kidney has undergone hydronephrotic dilatation, the kidney substance that remains being infiltrated here and there with masses of tubercle in different stages of caseation.

In some cases, in addition to the lesions produced by the tuberculous pyelitis, scattered masses of tuberculous deposit of varying size are found in the cortex, more especially at the junction between the cortex and the medulla.

The tuberculous deposit may in some cases undergo calcification, and it is not uncommon to find cretaceous tuberculous masses, both in cases of advanced renal tuberculosis, and also, but more rarely, as isolated masses in kidneys otherwise healthy, where death has resulted from tuberculous lesions in other organs. In a large proportion of cases of renal tuberculosis, the lower portion of the urinary or of the genito-urinary tract shows the presence of tubercle, and these deposits are more especially found in the testicles and in the prostate. In many cases, then, the affection of the kidney is really a secondary one, the primary tuberculous lesion being in the lower urinary tract, and the kidneys become attacked by direct infection passing up through the ureter, or else through the lymphatics in much the same way as in the so-called surgical kidney. In a considerable proportion of cases, the infection, however, is a descending one, the kidney being primarily affected and the lower urinary tract being infected secondarily from the kidney.

The evidence in favour of this is derived from the fact that, in some 40 per cent. of the cases of tuberculous disease of the genito-urinary tract, the kidney is the only part affected, whereas cases in which the disease is limited to the bladder or prostate are exceedingly rare, *i.e.* 2 per cent. Further, it is not uncommon in cases of tuberculous kidney for a tuberculous ulcer or deposit to be found at the entry of the ureter into the bladder, and cases are sometimes seen where a tuberculous mass undergoing caseation is found at one spot along the course of the ureter, often at a considerable distance from the kidney. It is difficult to understand these cases, on the view that the kidney lesion is secondary to the other, inasmuch as there is no direct continuity between the tuberculous lesion of the bladder or ureter and that in the kidney. On the other hand, if the renal lesion were primary, it can be readily understood how the ureter or bladder lesion might be induced by secondary infection, owing to the presence of bacilli in the urine. Although in many cases where tuberculous disease in the kidney and lower genito-urinary tract are associated, the renal lesion cannot be looked upon as secondary to the bladder lesion, yet in some cases this is obviously so, and the entire course of the ureter may be affected by the tuberculous disease from the bladder to the kidney, the ureter being thickened, and the mucous membrane not uncommonly ulcerated. In some cases the thickening of the mucous membrane of the ureter, from the presence of tuberculous deposits, is sufficient in amount to cause obstruction and pyonephrosis.

In cases where the kidneys are affected, secondarily to disease of the lower genito-urinary tract, both organs are commonly affected. In a certain proportion of cases the kidney is not only affected primarily, but the disease remains limited to the kidney. Inasmuch as the lower urinary tract may be affected secondarily from primary disease of the kidney, or primary disease in the lower genito-urinary tract may cause secondary infection of the kidney, the coexistence of the disease in the kidney and lower urinary tract is very frequently found clinically.

This close association between tuberculous disease in the genito-urinary tract and the kidney is only found in the male sex, and it is rare to find tuberculosis of the kidney complicating tuberculosis of the female genital organs. Even where the disease is originally limited to the kidney, as the malady progresses other organs become affected, more especially the lungs, the abdominal glands, and the intestine.

The disease is more common in men than in women, and although no age is exempt, it is most common in adult life before middle age.

Symptoms.—The involvement of the kidney in general miliary tuberculosis does not give rise to any symptoms. In cases where the kidney is involved by extensive primary disease, or where the organ is affected secondarily to disease of the lower urinary tract, symptoms are usually present, and sometimes are severe. The most constant effects produced by renal tuberculosis are the symptoms of a pyelitis, resembling in many points calculous pyelitis. In addition to the symptoms of pyelitis, in a considerable proportion of cases effects dependent on the presence of a renal tumour, such as pyonephrosis, are produced.

In addition to these two sets of effects, the general results of tuberculous disease are seen, such as wasting, pyrexia, and sweating. The pyelitis that is produced by tuberculous disease is characterised by frequency of micturition, marked equally by night as by day. The frequency of micturition in this disease is often extreme, rivalling that seen in cystitis, and perhaps, in some cases, is partly dependent on the infection of the lower urinary tract and of the trigone of the bladder, either primarily or secondarily. Pain is usually present, in the form of dull, continuous pain across the lumbar region, frequently unilateral and corresponding to the side which is affected. In addition to the constant lumbar pain, which in some cases is so severe as to rival that seen in caries of the spine, there may be attacks of renal colic, dependent on the passage of blood clot or broken-down fragments of tuberculous tissue.

Hæmaturia is commonly present, but is not necessarily continuous, and is usually slight in amount. It is exceptional in cases of tuberculous kidney for profuse hæmaturia to occur, but occasionally a profuse hæmaturia has been the first symptom of ill health.

Pus is always present in the urine, and it may be said that the characteristic urine secreted, in a case of tuberculous kidney, is one containing a considerable quantity of pus and only a small quantity of blood, whereas in calculous pyelitis it is more common for more severe hæmaturia to be present, together with a small amount of pus. The urine is usually acid, and it may contain small quantities of albumin in addition to that present, owing to the pus. Sometimes the amount of albumin is abundant, and is dependent on the supervention of waxy disease.

The presence of tubercle bacilli can often be demonstrated in the urine, either by staining or by inoculation. The physical signs of an enlarged kidney, dependent on a pyonephrosis, may be present, and sometimes it can be detected that the kidney is definitely enlarged on both sides.

With the progress of the disease the general effects of the tuberculous lesion become developed, great emaciation ensues, the hectic fever is marked, and sweating may be a troublesome feature. Pulmonary and intestinal complications, such as the development of phthisis, or of abdominal tubercle and tuberculous enteritis, are frequent. Death usually occurs as the direct result of the tuberculous process, the patient being worn out with exhaustion by the effects of the long-continued fever, or from the extension of the mischief to other organs.

In cases of bilateral tuberculous disease, death, however, may result from uræmia.

Diagnosis.—Most cases of tuberculous kidney present themselves in the form of cases of pyelitis, and the differential diagnosis has to be made from other varieties of pyelitis, especially calculous. Tuberculous pyelitis occurs more especially in young adults, and more frequently in men than in women. The pyuria is usually more marked, and the hæmaturia less marked than in cases of calculous disease. In a large number of cases the diagnosis has really to be made by the detection of tubercle bacilli in the urine. In other cases, where the symptoms of pyelitis are slight, the patient may come under observation for pain in the loin and the presence of a tumour. In such cases a differential diagnosis has to be made from retroperitoneal abscess, or even from psoas abscess, inasmuch as a pyonephrosis occurring as a result of the complete blocking of the ureter, may be unaccompanied by the presence of pus in the urine.

Where a tumour is present, together with pus in the urine, considerable assistance to diagnosis is afforded by the detection of tuberculous disease in the prostate or in the testicle. Enlargement and thickening of the ureter may be detected on vaginal examination, and will then greatly assist in the diagnosis.

Prognosis.—The prognosis of tuberculous kidney is very grave, owing to the frequency with which both kidneys are affected, and owing to the fact that, by the time the disease can be diagnosed, it has usually made great inroads. The duration of the disease is usually measured by months after the onset of symptoms, but some cases have lived for as long as three years.

Treatment.—In cases of unilateral tuberculous disease of the kidney, removal of the organ offers a very fair prospect of success, the main difficulty being the recognition of the fact that the disease is unilateral. In some cases examination of the bladder with the cystoscope and catheterisation of the ureters will reveal that the disease is bilateral, and in such cases operative interference can be of little or no avail. If there are, however, good reasons for thinking that the disease is unilateral, nephrectomy offers the best chance. Medical treatment can only be conducted on general principles for improving the general health, by fresh air, exercise, tonics, and by prescribing diuretics to freely dilute the urine.

Some benefit may perhaps be obtained by prescribing disinfectants, such as the salts of benzoic acid, or urotropin.

PYELITIS.

Etiology.—Inflammation of the pelvis of the kidney may exist by itself, but it may also be associated with inflammatory mischief, and especially suppuration of the substance of the kidney itself. To the latter affection the term pyelonephritis is frequently applied.

Pyelitis is generally dependent on the extension upwards of inflammation from the lower urinary tract. In these cases it is frequently accompanied by dilatation of the ureter and the pelvis of the kidney, since in many such cases there has been obstruction to the exit of urine. In other conditions pyelitis may exist as a more or less primary affection, and be independent of any inflammatory trouble of the lower urinary tract.

The presence of calculi and tuberculous growths in the pelvis of the kidney are two of the most common causes of pyelitis, and in both these conditions the inflammatory disease may be limited to the pelvis of the kidney.

Cystitis is frequently complicated by pyelitis, and in some such cases, as in the cystitis and pyelitis that may follow gonorrhœa, the inflammatory disease may pass up from the bladder to the pelvis of the kidney with little or no distension of the ureter. The cystitis which is associated with calculous disease of the bladder, with enlarged prostate, and with tumours of the bladder, is often complicated by pyelitis; but in such cases the disease usually supervenes as a late complication, and the long-continued bladder disease has caused dilatation and distension of the ureters.

Most cases of pyelitis are dependent on one or other of the above causes; in some cases, however, a primary pyelitis is seen as a complication of acute diseases, more especially pneumonia, typhoid fever, and scarlet fever. Here, in all probability, the infecting agent reaches the renal pelvis through the kidney, as it is well known that, in many diseases, and especially in typhoid fever, the urine contains a considerable number of bacilli. Certain irritant poisons, such as turpentine and cantharides, are also reputed to produce pyelitis, and in the case of the latter the nephritis produced at the same time may be so violent as to cause abscess of the kidney. Pyelitis is said to arise occasionally as a result of exposure to cold, but this is very doubtful; in almost all cases the disease is either dependent on irritation, produced in the pelvis of the kidney by calculi, tuberculous or other growths, parasites, *e.g.* bilharzia, or else the toxic agent reaches the pelvis of the kidney more usually from below, and the infection is an ascending one, but occasionally the infection reaches the pelvis of the kidney through the blood stream.

Morbid anatomy.—The changes presented by the kidney differ considerably in different cases, since pyelitis only may be present, or the pyelitis may be accompanied by other secondary changes, such as pyonephrosis and pyelonephritis.

In simple pyelitis the mucous membrane of the pelvis of the kidney is thickened and opaque, often greyish in colour, presenting here and there punctiform hæmorrhages of varying size; and in some cases, owing to the presence of necrosis of the epithelium, the site of these hæmorrhages may be left as ulcers. In calculous pyelitis the mucous membrane is not only thickened, but may be also villous in appearance. In some cases of pyelitis, more especially in those accompanying the acute specifics, an exudation forming a false membrane may be found coating the mucous membrane. The pelvis of the kidney will contain pus, and on section the mucous membrane will show the ordinary changes characteristic of inflammation—distension of the blood vessels, accumulation of leucocytes, etc.

In many cases, more especially where the pyelitis is consecutive to cystitis, the pelvis of the kidney and calyces will be dilated and the pyramids flattened. Areas of suppuration may often be found in the pyramidal portion of the kidney, extending up between the pyramids into the cortex, and associated with numerous small abscesses in the cortex just beneath the capsule. In some cases, owing to the confluence of neighbouring small abscesses, single large cavities containing pus may be found. Necrosis of the papillary portion of the pyramids may also occur, and occasionally necrotic masses of kidney substance are detached and voided through the ureters. To these conditions the name of surgical kidney or pyelonephritis is given, and the suppuration extends from the pelvis of the kidney to the cortex of the organ, owing to the organisms passing up the tubules, or frequently through the lymphatics, which lie in the interstitial tissue between the tubules. Where pyonephrosis is seen as a result of pyelitis, the kidney becomes distended into a sac containing pus, the wall of the sac being formed by the atrophied renal tissue. In such cases the wall of the abscess differs somewhat from the wall of a hydronephrosis, owing to the considerable formation of fibrous tissue and the greater development of necrotic changes in the renal cells.

In cases of consecutive pyelitis the infecting agent ascends from the bladder either along the lumen of the ureter, or else through the lymphatics. Distension of the ureter, from obstruction to the exit of urine, necessarily is a powerful disposing cause to the onset of pyelitis.

Some cases of pyonephrosis are seen coming on suddenly in healthy young persons, without any previous history of pyelitis, and where no stone may be found on operation. In such cases the pyelitis has probably been produced by infection reaching the kidney through the blood, and the pyelitis has produced obstruction to the ureter, so causing the pyonephrosis.

Symptoms.—The symptoms of pyelitis will vary a good deal, according to whether the disease exists by itself, or is complicated by the presence of pyelonephritis or by pyonephrosis.

In simple pyelitis, pain, frequency of micturition, and the changes produced in the urine by the presence of inflammatory mischief, are the most characteristic effects. The urine is acid, and contains a variable amount of pus. Sometimes the presence of pus in the urine is transitory, owing to the ureter of the affected side becoming blocked transitorily. Hæmaturia may be absent; at other times the urine is smoky, but it does not as a rule contain large quantities of blood unless the pyelitis is chronic and dependent on calculous disease, and then profuse hæmorrhage may occur from time to time. In such cases the mucous membrane of the renal pelvis may be villous or even ulcerated. In cases of simple pyelitis the amount of albumin in the urine is small, and is merely dependent on the presence of pus. Amyloid disease of the kidney may exist as a complication of the pyelitis, and in such cases the amount of albumin present is much larger than can be accounted for by the mere pyuria. Fever and rigors are usually present, more especially in cases of acute pyelitis; and the temperature chart presents very great variations. The febrile paroxysms have been mistaken for those of malaria, owing to their recurring at more or less regular intervals. In cases of long-continued pyelitis, whatever their origin may be, the fever assumes the hectic type.

The general condition of the patient will depend a good deal on the duration of the pyelitis, on its causation, and more especially on whether such complications as pyonephrosis and pyelonephritis are present. In cases of simple pyelitis the general health need not suffer to any great extent, unless the fever is very high and the disease long-continued; but where, as is so often the case, pyelonephritis is also present, emaciation becomes a marked symptom; and such patients are not only wasted, but are pale and sallow, and have a cachectic appearance, very similar to that seen in malignant disease. Often in such cases the temperature is not very high, not above 101° F., but the patient is profoundly ill, suffering from frequent rigors, and the tongue becomes dry, brown, and cracked; uræmic symptoms, such as nausea, vomiting, cramps, and twitchings soon develop. In cases where pyonephrosis is present, the patient may suffer from all the effects associated with the presence of a deep-seated abscess; the pain is frequently severe, the temperature high, and on an abdominal examination the tumour is readily detected.

Pyelitis may be unilateral or bilateral; in calculous disease it is often, and in tubercle it is occasionally, unilateral. Where it occurs as a sequel of cystitis, it is commonly bilateral. Pyonephrosis, as mentioned above, is a frequent complication of pyelitis, and it may lead to other complications itself; thus the pus contained inside the distended pelvis may burrow through the origin and form a retroperitoneal abscess, and this, if not recognised, may burrow widely in various directions, and more especially upwards, forming a subphrenic abscess, which may perforate the diaphragm, and so lead to empyema or to abscess of the lung. In other cases, if the pyonephrosis or retroperitoneal abscess is not detected, a general pyæmia may result. In some cases of pyelitis complicated by pyelonephritis, abscess of the kidney occurs as a complication, and is generally dependent on the confluence of several small abscesses.

Abscess of the kidney, although frequently the result of pyelitis, may occur as a result of injury to the loin or as part of a general pyæmia, in ulcerative endocarditis, or as a complication of the presence of a calculus in the kidney. The presence of abscess of the kidney produces the general symptoms associated with pus formation, such as rigors. Hæmaturia and pyuria may be present, but pyuria is often absent throughout the illness; the more usual and typical course is for a patient suffering from irregular fever to suddenly discharge a considerable quantity of pus in the urine, and for the temperature to suddenly subside. In other cases the abscess enlarges in such a manner as to burst through the capsule of the kidney, and to form a retroperitoneal abscess, which may often increase in size to a sufficient extent to be detected clinically.

Diagnosis.—The diagnosis of pyelitis is usually made upon examination of the urine, and the condition is always to be suspected when pus is present in acid urine, more especially if tenderness on pressure is found in one or both loins, or if pain in one kidney is a marked feature. In the pyelitis, however, following cystitis, the urine may be alkaline, and in some cases of cystitis, dependent on the *Bacillus coli communis*, pus may be present in small quantities in the urine without the acid reaction disappearing. In most cases the diagnosis is based on the combination of the presence of pus in the urine, together with the signs pointing to the affection of one or both kidneys. It is often difficult to determine whether pyelitis alone is present, or whether the disease is complicated by pyelonephritis. In the latter condition the patient is more wasted, more ill, suffers from a recurrence of rigors, and the urine is often of low specific gravity, and may be secreted in abundance.

The main difficulty in the diagnosis of pyonephrosis lies in the fact that it is liable to be confounded with other collections of pus in the abdomen, and more especially with retroperitoneal abscess. In pyonephrosis the urine frequently contains no pus; in fact, in the majority of cases, it does not, owing to the occlusion of the ureter being complete; and so the differential diagnosis has to be made rather on the signs of a renal tumour, together with the presence of fever, than on results deduced from an examination of the urine.

Prognosis.—The prognosis of pyelitis depends on its cause, and on whether the disease is complicated by pyelonephritis. In the latter case the outlook is exceedingly grave. The prognosis of tuberculous pyelitis and of the pyelitis that is the sequel to the obstruction in the lower urinary tract, is also grave. On the other hand, pyelitis, the result of the presence of calculi, may last for many years, and with suitable treatment of the cause may disappear. Pyelitis following such a condition as gonorrhœa is sometimes fatal; but in other cases, even where it has caused pyonephrosis, it yields to operative treatment. The pyelitis seen in the course of acute specific fevers is usually a trivial affection, and subsides with the disease causing it, although it may lay the foundations of other diseases as a sequel. Pyelitis arising in the first instance as the result of an acute specific fever, and so trivial as not to be recognised at the time, may lead to the formation of calculi, the blood or pus influencing the mode of crystallisation of uric acid or of oxalate of lime, and so forming the nucleus of a calculus.

Treatment.—Many varieties of pyelitis, especially the calculous, require surgical interference, and this is also necessary where the disease is complicated by the presence of pyonephrosis. In other cases the patient should be kept in bed, if the disease is acute, and fed on a light diet—chicken, fish, milk, and farinaceous food. The patient should take considerable quantities of simple drinks, such as barley water, linseed tea, buchu, in order to freely dilute the urine; and acetate of potash, in doses of from 20 grs. to a drachm, freely diluted, is often of use. Stimulants should be avoided. If the urine is alkaline, benzoate of ammonia may be prescribed, with the view of rendering it acid; and recently urotropin, in doses of 10 grs., has been recommended as a mild urinary antiseptic. Quinine is often useful for the treatment of the fever. If cystitis coexists, and is the cause of the pyelitis, this will necessarily require the usual treatment.

Pyonephrosis requires surgical treatment, either in the form of draining the distended kidney, or else in some cases nephrectomy. In cases where the disease arises as a result of obstruction of the ureter, *e.g.* calculus, and this is found and removed at the operation, drainage of the abscess cavity is often sufficient. In other cases where the obstruction is not found, or cannot be removed, mere drainage is apt to be followed by the persistence of a sinus discharging pus and decomposed urine, and not uncommonly this condition necessitates a second operation to remove the remains of the kidney.

NEPHROLITHIASIS.

The formation of calculi or gravel in the kidney is associated with either some general disturbance of the metabolism of the body, leading to an alteration in the composition of the urine, or else with some morbid process in the pelvis of the kidney, owing to which some constituents of the normal urine are deposited in an anomalous solid form.

Renal calculi are most frequently formed of uric acid or of oxalate of lime. Some of the other and less abundant constituents of urine give rise to the formation of stones under certain circumstances, as for instance carbonate of lime, which in at least two cases has been found as the main constituent of gravel formed in the kidney. Cystin, a substance which is present normally in very small quantities in the urine, may also form a calculus, and concretions consisting of xanthin have been described in very rare instances.

Amongst the rare forms of renal calculi are those consisting of carbonate of lime and of basic phosphate of lime. The former, carbonate of lime, may occur either in the form of gravel or else very exceptionally forms a single large branched calculus.

Calculi consisting of the mixed phosphates—the so-called fusible calculi—are not uncommon in the bladder, as a secondary deposit, round nuclei formed usually of uric acid or of oxalate of lime. In very rare instances calculi consisting of indigo have been described. In many of these cases the ingredient forming the calculus is one that exists normally in the urine, and can be deposited from that fluid without its undergoing any decomposition, *e.g.* uric acid. In other cases the deposit can only occur as a result of the occurrence of fermentation and decomposition of the urine, so that its reaction is alkaline from the presence of ammonia. This is especially the case where the calculus consists of the triple phosphate. For this reason this calculus is generally found, not in the kidney but in the urinary bladder.

Calculi are formed most frequently in the pelvis of the kidney, but sometimes they are formed in a very minute form in the tubules. When, as is generally the case, they are formed in the pelvis of the kidney, they may be single or multiple, in some cases several hundreds being present. When single they may reach a large size, and branch so as to form a cast of the pelvis and of the calyces of the kidney, and in such cases they may reach a very large size. It is not uncommon for stones to be facetted, and to be accurately fitted one against another. Some renal stones are smooth and rounded, as in the case of some uric acid calculi, others are studded with minute prominences or with facets. The oxalate of lime calculus has usually a tuberculated surface, from which it derives its name of the mulberry calculus. Calculi consisting of carbonate of lime are often small in size and spherical in shape, but, as mentioned above, they may be large and branching.

In some cases the composition of a renal stone is fairly uniform, consisting almost entirely of the one ingredient, uric acid, oxalate of lime, etc.; in other cases the composition is mixed, forming the so-called alternating calculus. In children, calculi, or at any rate the nuclei of calculi, may be formed of urate of soda, or of urate of ammonium.

Etiology.—Calculi may be formed at all ages, but they occur very frequently in childhood; and it is usually said that stones occurring in childhood are most likely to contain ammonium or sodium urate; those in young persons, uric acid; and those after middle life, oxalate of lime. This difference in the composition, however, applies more especially to the nucleus. Alternating calculi, consisting of alternate layers of uric acid, oxalate of lime, or of phosphate of lime, are very common. The composition of the calculus is necessarily associated with the reaction of the urine. If the urine be persistently acid, the stone will probably consist of either uric acid or of oxalate of lime. If the urine be alkaline, from the presence of fixed alkalies, the rare stone formed of carbonate of lime or basic phosphate of lime may be formed; and it is only in the case of alkalinity from the presence of a volatile alkali that the calculus consisting of the triple phosphate will be formed.

Uric acid calculus.—The formation of a uric acid calculus is intimately associated with the conditions which determine the deposition of free uric acid from the urine. All urines, if prevented from decomposition, deposit uric acid sooner or later, but in most cases this deposition does not occur until many hours, *e.g.* twenty-four to forty-eight, after the passage of the urine. In some cases, however, it occurs very rapidly after emission, and if it occurs before this the conditions for the formation of stone may be present. Although the amount of uric acid present in the urine to a certain extent influences the time of its deposition, more important factors are the reaction of the urine and the percentage of saline material, especially phosphates, present. A high acidity of urine, together with a scanty amount of saline ingredient, will favour the precipitation of uric acid, as the uric acid is normally kept in solution in the urine, thanks to an interaction between the urates and the phosphates. Some observers consider that the uric acid is normally present in the urine, in the form of a soluble salt known as quadriurate; others look upon the quadriurates as a mixture of a biurate and free uric acid; but all are agreed that the formation of the hypothetical quadriurate or of a biurate is intimately associated with the presence and amount of the other salts of the urine, more especially the phosphates.

Highly concentrated urines containing an abundance of urates, as for instance in febrile processes, do not deposit uric acid freely, owing to the large amounts of other salts present. On the other hand, the dilute urine that is secreted in the granular kidney, and in other conditions, is one where, although the percentage of uric acid is less, the tendency to deposition is greater, from the lack of other saline ingredients. In the case of uric acid calculi, the deposition of the material is more closely associated with the chemical composition of the urine than in the case of the oxalate of lime calculus.

Oxalate of lime calculus.—In this condition the most important factor that we are acquainted with, influencing the formation of oxalate of lime gravel, or oxalate of lime calculus, is the amount of oxalate of lime present in the urine. This is largely influenced by diet, many articles in common use tending to increase the amount of oxalate of lime in the urine, but more especially rhubarb, tomatoes, sorrel, gooseberries, beans, etc. Some patients, however, with the so-called oxalate of lime diathesis, will pass considerable quantities of this salt in the urine, apart from the ingestion of these substances.

Determining causes of calculus.—The mere presence of the particular substance which leads to the formation of a calculus in excessive amounts in the urine, is not generally sufficient to account for the formation of stone. There must be, generally, some determining cause acting locally on the kidney. This, in the majority of cases, is probably some comparatively slight inflammatory affection of the mucous membrane of the renal pelvis, leading to the secretion of an excessive amount of mucus, as in catarrhal pyelitis, or else a congestion, leading to the exudation of some albumin, or perhaps, in many cases, to a small hæmorrhage. The presence of this colloid material in the urine may then so influence the form of crystallisation of the uric acid, oxalate of lime, or other substance, that either an abnormally large crystal is formed, or else a deposit occurs of the uric acid, urates, or oxalate of lime in a globular form, and this acts as a nucleus for the subsequent growth of the calculus by accretion. In many cases the exudation of this colloid material into the urinary passages may occur as a result of a slight pyelitis, occurring in the course of an acute disease, and too trivial to attract notice at the time. It is probable that some local condition of this kind plays a great part in the production of renal calculus, and may afford a ready explanation of the fact that the disease is usually limited to one kidney. Any generally acting cause, such as an increased amount of uric acid in the urine, or an alteration in the reaction of the urine, would operate equally on both kidneys. Although calculi are found in some 15 to 20 per cent. of cases on both sides, yet in the great majority of cases they are limited to one kidney.

The etiology, therefore, of renal calculus may be summed up as, in part, dependent on metabolic disturbances affecting the composition of the urine, in the manner described above; and, in the second place, some local condition of pyelitis determines the actual formation of the calculus and the particular side affected.

Diet and habits, therefore, will play a large part in the etiology, especially of the uric acid calculus; and in many such cases the disease can be traced to the habit of eating very large quantities of meat. This will operate in two ways—first of all, in increasing the acidity of the urine, owing to the larger amounts of acid phosphate and soda ingested and excreted; and, secondly, by increasing the amount of uric acid. A diet poor in salts may also tend to the formation of uric acid calculi, not by increasing the amount of uric acid present, but by withholding the necessary amount of saline material to keep the uric acid in the soluble form of the quadriurate or biurate. This may account for the prevalence of uric acid stones amongst the poor, and also in such countries as India and China, where rice forms the staple article of diet, and rice is a material very poor in saline ingredients. Climatic and telluric conditions may possibly play a part; thus stone is very prevalent in Norfolk, and it is probable that the condition is really dependent on the amount of saline material in the water drunk.

Sedentary habits probably act as a predisposing cause. Stone is more common in men than in women, in the proportion of five to one.

Morbid anatomy.—The presence of a stone in the pelvis of the kidney may produce no effects on the kidney; more usually, however, it causes pyelitis, and in some cases it leads to ulceration of the mucous membrane of the pelvis. The most common secondary effects produced on the kidney are atrophy and hydronephrosis, in some instances pyonephrosis and pyelonephritis, and in a small proportion of cases the long-continued presence of calculi in the kidney has been associated with the subsequent development of cancer in that organ. In cases of atrophy the kidney sometimes remains as a shrivelled sac, retaining few, if any, signs of its structure; and in such cases it is probable that a hydronephrosis has been first of all produced, and that this has become emptied, and the kidney has then atrophied. In other cases, however, genuine atrophy is seen where the organ retains its former and normal shape, but has become dwindled in size and indurated from the overgrowth of fibrous tissue. In a considerable number of cases the presence of calculi in the pelvis leads to the complete suppression of the secretion of the urine, without producing any very marked anatomical changes in the kidney, during the short time (seven to twelve days) that life persists, and in some such cases the suppression is not associated with the presence of any pent-up urine in the renal pelvis, true suppression being produced.

Although hydronephrosis and pyonephrosis of large size may result from the impaction of a renal calculus in the ureter or pelvis of the kidney, in some cases the distension of the kidney is moderate in amount, owing to an enormous overgrowth of fibrous tissue in the kidney and in its capsule, from the presence of chronic perinephritis.

Symptoms.—In a considerable proportion of cases, the presence of stones in the kidney produces no symptoms, and they are only discovered accidentally in the post-mortem room. In many such cases the stones are of large size, and branched, ramifying and following the pelvis and calyces of the kidney. In other cases numerous small stones may be present, and may often be passed from time to time without the production of any symptoms. More usually, however, when the stones are passed, the phenomena of renal colic are produced. In some cases the presence of calculi gives rise to no symptoms until complete suppression suddenly occurs. In such cases the usual condition is for one efficient kidney only to be present, the other being congenitally absent, or having undergone atrophy in early life, perhaps as a sequel to calculous disease, or to other causes, and the suppression is produced by the distinction of the ureter of the single efficient kidney. In some cases of complete suppression, both ureters are simultaneously obstructed by the presence of calculi; this, however, is rare.

Where complete suppression of urine occurs, as a result of impaction of a calculus in the ureter, it is more common for the patient previously to have presented symptoms, pointing to the presence of calculous disease; but, unquestionably, cases occur where, as just mentioned, the occurrence of complete suppression is the first sign pointing to the existence of this disease.

In other cases, hydronephrosis or pyonephrosis of the ordinary character are the initial effects produced by the stone.

In the majority of cases presenting symptoms, however, the presence of stone in the kidney gives rise to attacks of renal colic. The attacks of pain in renal colic may take one of two forms. In the first, the pain is limited to the loin, and to the anterior surface of the abdomen in the hypochondriac and lumbar region, and is confined to the side on which the stone is present. In the second variety, the pain, after being felt in this situation, extends in a downward direction obliquely towards the scrotum and groin, and in some cases extends a considerable distance down the inner side of the thigh. The pain is commonly very severe, and the patient frequently rolls in agony on the ground. It is also very commonly accompanied by tenderness of the skin of the loin and of the anterior surface of the abdomen. The cutaneous tenderness extends commonly in a band-shaped area, passing from the twelfth dorsal to the third lumbar spine behind, and extends more or less horizontally round the body to the middle line anteriorly, and forms a band of hyperæsthesia between the umbilicus and the symphysis pubis. Where the pain extends down into the groin and into the scrotum, it is accompanied also by cutaneous hyperæsthesia in these situations. During the attack of pain, there is not only superficial tenderness of the skin of the abdomen, but there is also deep tenderness over the region of the kidney, so that palpation of the abdomen is painful. During the paroxysm of the pain the abdominal muscles are rigid and board-like. In exceptional cases, the pain not only radiates downwards into the groin and into the scrotum, but also spreads down the back of the thigh, and in some cases it may spread as far as the heel; this is very exceptional. The pain in the back, in the loin, and in the abdomen is frequently more constant than the radiating pain into the scrotum and groin, and the latter is apt to occur in paroxysms, in the intervals the pain passing away entirely. The former is more apt to be persistent, and for exacerbations to occur from time to time.

The duration of the pain is very uncertain; in most cases it lasts for several hours with temporary remissions, and in the form in which the pain radiates down to the groin and the scrotum the relief experienced is sometimes quite sudden. After the subsidence of the pain, soreness and aching persist in the loin and in the back always, for some hours, and sometimes for days. The attack of pain may come on with extreme suddenness, occasionally after some jolting, but in other cases without any predisposing cause, and even in the middle of the night. Where the pain radiates down to the scrotum and the groin, the stone has entered the ureter; and this variety of pain is unquestionably associated with the passage of the stone along the ureter, and the sudden relief of the pain may be dependent on the stone slipping into the bladder. It is said that in some cases the patient will actually experience that sensation. Where the stone is large, or from some other cause is unable to enter the ureter, the attacks of pain are limited to the lumbar region. It is said that the pain may be sometimes on the opposite side to that on which the stone is present.

The pain associated with calculus, although more severe than that seen in other renal affections, does not differ absolutely from the pain that is seen in some cases of movable kidney, or which is seen as the result of the passage of blood clots or fragments of renal tissue or growths.

The onset of renal colic is often accompanied by a rigor. The temperature may rise to 103° F., or even higher. In many cases the occurrence of rigor is associated with pyelitis or other inflammatory complications, but renal colic, like biliary colic, may be accompanied by rigor, without the presence of any marked inflammatory complication.

Nausea and faintness are usually present, and the patient becomes pale and covered with a clammy sweat, and in severe cases there may be a considerable amount of collapse. Vomiting usually occurs; in some cases the vomiting is such a marked symptom as to simulate, by its violence, some intestinal trouble, such as obstruction.

During the attack of pain, frequent micturition and even tenesmus may be present; the flow of urine is diminished, and the urine that is passed is scanty, high-coloured, and commonly smoky. In some cases considerable quantities of blood may be passed, but it is more usual for the hæmaturia to be moderate in amount. In some cases the flow of urine is completely suppressed during the paroxysm of pain, and in cases where calculi become simultaneously impacted in both ureters, or where, owing to previous disease, calculous or other, one kidney is functionless, the suppression may be persistent and fatal.

Where the disease is of long standing, and the presence of the stones has set up pyelitis, the urine will of necessity contain pus. Calculous anuria produces a remarkable train of symptoms, in which, notwithstanding the fact that the patient may pass no urine, life may be prolonged for a week or ten days, or even, in some cases, twelve. In such cases, notwithstanding the complete suppression of the urinary secretion, the patient remains conscious and frequently perfectly rational to within a few hours, or even to as short a period as half an hour, from the time of death. They complain of few symptoms, with the exception of suffering from extreme lassitude and weakness, and after the condition has persisted for five or six days, slight nausea and eructations may be present. The temperature becomes subnormal, falling to 95° F.; the pupils become contracted, and the knee-jerks are commonly exaggerated. After the condition has persisted for a week or so, slight twitchings of the muscles, especially the extensors of the forearm, may be detected, and the respiration may be slightly embarrassed. Delirium, coma, and convulsions, the ordinary accompaniments of uræmia, are, however, absent. Death occurs commonly with great suddenness, from respiratory failure. In some cases slight œdema may be present, but this is not a constant phenomenon, and very often, when it is present, it disappears before death.

The clinical picture produced by suppression of urine in calculous obstruction is very definite, and has been described as that characteristic of obstructive suppression, as it is seen not only in cases of calculous obstruction but also in other conditions, where the ureters are obstructed by malignant or other tumours of the pelvis. The condition was originally described by Sir William Roberts, and the distinctions between it and ordinary uræmia, supervening in acute and chronic renal disease, emphasised. Although it is very characteristic of calculous and other diseases obstructing the flow of urine, it is not really limited to these, and precisely the same group of symptoms may be seen in cases where the functions of the kidneys, up to that time healthy, are suddenly suppressed by vascular lesions obstructing the renal arteries, or, in some cases, by the actions of the toxines of such diseases as diphtheria. In obstruction of the renal arteries, entailing complete necrosis of both kidneys, with complete suppression of urine, the patient may live for a week, as in cases of calculous obstruction, and present identical symptoms. The term, latent uræmia, may be perhaps used to describe the group of symptoms described above as supervening in these cases, and it is certainly remarkable that the functions of the kidney in the human subject may be entirely abrogated, without the onset of the ordinary phenomena characteristic of acute uræmia. It is a point of some importance, as regards the diagnosis of calculous obstruction, that the condition of latent uræmia is not really limited to this condition, but may occur in diseases in which the kidneys are entirely and irretrievably damaged or destroyed by acute vascular lesions, or even, in rare cases, where suppression occurs as a reflex phenomenon, produced by disease elsewhere, *e.g.* perforative peritonitis.

Diagnosis.—The diagnosis of calculous disease may be based, either on the occurrence of renal colic, or else on the presence of pyonephrosis, pyelitis, or even the sudden onset of so-called obstructive suppression, with latent uræmia. If attacks of renal colic are present, the disease has to be diagnosed from biliary colic and from intestinal colic. The differential diagnosis is usually made by a careful consideration of the area and distribution of the pain, together with the presence of tenderness over one kidney, and the occurrence of hæmaturia. The region of the pain in biliary colic is quite different to that in renal colic, the one tending to pass downwards towards the scrotum, the other upwards towards the shoulder and neck. In intestinal colic, rigors are usually absent, but they may be present in biliary colic.

Attacks of pain may occur in movable kidney which simulate renal colic, and cases of so-called renal neuralgia have been described, where attacks of pain, indistinguishable from those of renal colic, have occurred, but where exploration, and even removal, of the kidney have failed to show the presence of a stone.

Prognosis.—The prognosis of calculous disease is very uncertain, both from the point of view of the recurrence of the attacks of pain, and also as regards the development of such complications as pyelitis, pyonephrosis, calculous suppression, or even malignant disease. Suitable treatment, however, medical or surgical, will, in a considerable number of cases, where the kidneys are not seriously damaged, not only lead to relief, but even to the cure of the affection.

Treatment.—If the patient presents distinct evidence of the presence of a stone in the kidney, it is probable that medical treatment is quite unavailable to effect either the absorption of the stone, or, if of large size, even its expulsion. Large stones, causing only the first variety of renal colic where the pain does not radiate, cannot be passed by the ureter; and in such cases nephrotomy affords the only possibility of relief, if the attacks of pain are of sufficient severity to incapacitate the patient from his occupation or work, or where pyelitis or pyonephrosis has developed as a complication. In attacks of renal colic, temporary relief is obtained by hot applications to the loin, and a hot bath will very often relieve the spasm of the ureter, which gives rise to the pain. Where this fails, and the pain is very severe, morphine should be employed, or even chloroform. It is said that inversion of the patient will sometimes cause immediate relief. The diet of a patient suffering from renal calculus should be very similar to that recommended in the treatment of gout, and in cases of uric acid calculi, dietetic treatment should be directed towards the rendering the urine less acid, by diminishing the amount of meat taken, and increasing the vegetable diet. This, however, might not be suitable in cases of oxalate of lime calculus. Citrate of potash may be prescribed in drachm doses at night, in order to render the morning urine less acid, but it is impossible, by the use of such treatment, to dissolve stones that have been formed, although such treatment may prevent the formation of fresh stones. To effect this, the acidity of the urine must be diminished, or the urine must be rendered alkaline, and it should also be abundant, and therefore the patient should be recommended to drink largely of simple mineral waters or of ordinary plain water. Such springs as Vichy, Ems, Contrexéville, Wildungen, are also useful in the treatment of these cases.

JOHN ROSE BRADFORD.

SECTION IX.

INTEGUMENTARY SYSTEM.

INTRODUCTORY.

If due regard be paid to the importance of a healthy skin for the well-being of the economy, the consideration of the disorders to which the integument is liable may, in time, be estimated at its true value. Many of these seriously cripple, or altogether interrupt, the power of earning a livelihood; others disfigure to such a degree as to render exposure a severe trial; some, though concealed from public gaze by being localised on covered parts, yet are the seat of more or less constant itching or annoyance; while a certain number tend slowly, though surely, to a fatal issue. A large majority are curable, especially if attacked early; while most, if not all, are capable of great amelioration by suitable treatment. The rise of dermatology is almost coincident with the Victorian era. Though a foundation had been laid by British and French observers before his time, yet it is to Hebra, who commenced his classic writings in the year 1844, that we owe its establishment on a scientific basis. Many of his statements remain unchallenged even now; others, indeed, have undergone revision or modification; but the impetus he imparted to the study of cutaneous disease has never once ceased to operate.

While skin diseases enjoy the advantage of being visible, yet the almost infinite varieties of form, colour, and appearance which they assume render their exact determination in many cases no easy task. This has no doubt been facilitated by the many beautiful pictorial representations in the numerous atlases available, but this assistance has been lessened by the puzzling and multiple nomenclature. Several names have at different times been attached to the same disease, making identification, when an atlas is referred to, difficult. In examining any skin affection, it is desirable that the entire surface of the body should be inspected in a good light, daylight if possible, so as to compare the appearances on diverse parts. Inquiry as to previous treatment should be made; but, ere a definite opinion is arrived at, the surface should be cleansed by a bath, and all accretions removed. It is well to avoid listening to any information volunteered, and to reserve questions as to history, till after inspection.

The characteristics, from the presence of which, singly or combined, the determination is made, have been divided into objective or apparent, and subjective or those perceived by the individual. The objective symptoms again are divided into primary and secondary lesions. The primary consist of—The macule—any abnormal change in the colour of the skin confined to a limited area; the papule—any morbid change in the skin which forms a solid projection above its surface, from the size of a millet seed to that of a lentil, and, so far as can be appreciated by the eye, containing no fluid; the vesicle—an elevation of the horny layer of the epidermis by transparent or milky fluid; the pustule—a small abscess, covered only by epidermis; the wheal—a solid induration, slightly raised above the surface, the superficial area greatly exceeding the thickness, accompanied by tingling or itching sensations; the tubercle—a solid, though not always hard, elevation, varying in size from a split pea to a cherry. The secondary are—Crusts—accumulations of exuded material and epidermis; scales—branny dust or flakes, more or less attached; excoriations—superficial breaches of continuity, due to scratching or friction; fissures—cracks in the epidermis, or reaching into the corium; ulcers—erosion of a portion of true skin; scars—connective tissue repair of a gap in continuity; pigmentation—brownish or dark staining, usually due to long-continued irritation; leathery induration—indolent thickening from partial organisation of plastic material. The subjective symptoms consist of various abnormal sensations, as pain, tenderness, heat, itching, formication or creeping, tingling, pricking, hyper- and anaesthesia.

There is a popular idea that cutaneous disorders are chiefly caused by dirt. This has a certain scientific basis, inasmuch as a number have been traced to parasitic or micro-organismal agencies; but the most scrupulous cleanliness is not incompatible with their presence. Nor are ablutionary measures alone capable of curing them; on the contrary, too much washing with soap often aggravates, sometimes directly induces, some. Arsenic, which has acquired the reputation of being "good for the skin and complexion," is only of value in a very select few; is rather prejudicial than otherwise in most, in the number of which eczema may be included. It, like mercury, should never be indiscriminately prescribed.

No thoroughly satisfactory classification of skin diseases has yet been formulated. The one employed here is that of Radcliffe Crocker, slightly modified. By it a division is made into six classes, as follows:—Diseases connected with the appendages of the skin; disorders presenting more or less of the inflammatory type; diseases in which the most prominent feature is overgrowth of one or more of the structures which constitute the skin; diseases characterised by the formation in, or infiltration of, the skin by new elements; disturbances of sensibility of the skin; parasitic diseases. This enables us to group together allied elements, but one class includes members which might perhaps equally well have been relegated to another. Some degree of overlapping is unavoidable at present, possibly will always be so.

The foreign ailments not discussed in this section are pellagra, Morvan's disease, ainhum, leprosy, yaws, and verruga peruviana. There are also eruptions in syringomyelia; perforating ulcer of the foot chiefly occurs in the course of leprosy, locomotor ataxy, and diabetes, and is referred to under these sections. Purpura, which is a symptom rather than a disease, has separate notice.

THE SEBACEOUS GLANDULAR APPARATUS.

SEBORRHŒA.

Excessive production of oil, appearing on the surface either as an oily coating or in scale-like masses, unaccompanied by visible symptoms of inflammation. It would appear that Rayer was the first to speak of a definite sebaceous flux; but Hebra, adopting the anatomical views of Virchow, drew the picture generally accepted. From this Unna dissents, for he regards the sweat glands as the true lubricants of the integument, their secretion being, in his opinion, an oily emulsion, the fatty portion of which emanates from the coil, while the watery is drawn from the tissues through which the long excretory duct passes. Though it is pretty commonly admitted that the sweat does contain fat, still Unna's views have not been wholly indorsed.

The sebaceous glands may be said to form part of the hair sacs, and their secretion is discharged partly by the contraction of the arrectores capillorum muscles, through apertures common to both structures. Their primary function is to supply oil to the hairs, and it is probable, as suggested by Schütz, that the secretion, normally fluid at the temperature of the body, is to some extent drawn from its source by capillary attraction. That this cannot be their entire office is shown by the fact that the bulk of the sebaceous glands is often, as in the instance of the lanugo hairs, quite out of proportion to their size. Such glands also, or their analogues, occur apart from hairs, as on the lips, the labia, and the prepuce; hence they must have a wider function.

Etiology.—The exciting causes of seborrhœa are probably organismal, several varieties being at work. Its apparent transmissibility, and its prevalence among those in vigorous health, favour this view. Unna claims that his morococci—surface micro-organisms appearing as diplococci, or grouping like mulberries—are the efficient agents. The condition of the soil must have an influence, since in so many cases is there coexisting anæmia, for this factor to be overlooked. In early syphilis it is coeval with the bloodlessness, and is common in convalescence from zymotic or inflammatory diseases. In these, however, it usually spontaneously disappears when health returns, and the due proportion of red corpuscles and hæmoglobin is restored. The intemperate use of alcohol is in some a cause of oily seborrhœa of the face, while the habit of wearing hard hats, by keeping the scalp too warm and occasioning local excess of perspiration, promotes it on the head.

Pathology.—This has been the subject of much discussion. Unna views all the varieties as inflammatory, though the clinical evidence in support of this is not always forthcoming. He explains it by chemiotaxis, or the attractive influence exerted by micro-organisms or their toxines on the mobile elements of the tissues. In this case it takes the form of a flux of fat, a sebotaxis. Sabouraud confirms the observations of Unna and Hodara as to seborrhœa being due to a special microbacillus, but holds that the implantation of this in the pilo-sebaceous follicle gives rise to sebaceous hypersecretion as an oily flux; to hypertrophy of the gland itself; to a progressive atrophy of the hair papilla, followed by death of the hair. Since, however, the micro-organism never penetrates so far as the root of the hair, he explains the destructive action as caused by toxines.

The organism continues its stimulating effect after the destruction of the hair, and the production of baldness, accounting, according to him, for the greasiness of bald regions. There is, however, another explanation, that the gland still pours forth its secretions, but this can no longer be absorbed by the hair. The scales and crusts found on a scalp affected with seborrhœa are due to other superadded microbes.

Symptoms.—The most typical excess of sebaceous flux is that in which it is persistently too fluid. Under favourable circumstances, we can see minute droplets of oil exuding from the dilated gland orifices on the nose. Sometimes also in women, their long hair, after being washed, rapidly reacquires a preternatural greasiness. The face looks shiny, and, from floating particles of dust becoming adherent to its surface, it presents a greasy, unwashed appearance. In consequence the skin may acquire a coarse texture, over which dilated venules may ramify. Though met with as a solitary condition, it is not infrequent in association with other cutaneous disorders in which the glandular system is implicated. It is undoubtedly to some extent dependent on constitutional disturbances. The state of the digestive and excretory functions affect it, and a tendency to it is sometimes left behind by an attack of erysipelas or variola. The integument is frequently pallid and inert.

The other variety of seborrhœa is a more complex ailment. In it there are more or less abundant greasy accumulations on the surface, not purely composed of fat, but consisting also of epidermic scales impregnated with oily material. Its most customary situation is on the scalp, where it may be found at all ages; and, while sometimes productive of little harm, it is the chief cause of premature baldness, while it predisposes to, or complicates, many forms of skin disease on all parts of the body. It is this widespread influence which it exerts which gives it so prominent a place.

In infants this greasy coating is met with, in particular, in the neighbourhood of the anterior fontanelle, where it attains some thickness, and, adhering firmly, it forms a dirty yellowish or blackish investment. Even if removed, it is apt rapidly to reaccumulate; but the efforts made to detach it are not always energetic, owing to a prejudice against interfering too much with that region.

In adolescence and full age, it is pretty extensively diffused over the scalp, with a predilection for the temples and crown, and occurs either as yellowish branlike particles closely set round the hairs, or as glistening white, hard, micaceous scales, which, dislodged in brushing the hair, fall in showers over the clothes, the condition being that known as dandriff. In the uncomplicated form the scalp beneath is pale, seldom at all reddened. A degree of itchiness often accompanies it. The drier variety is that most apt to lead to loss of hair. The masses of scales interfere with due lubrication, the hair becomes lustreless and brittle, falls off in excessive quantity, while that which replaces it is constantly thinner and scantier. Unna is of opinion that the withering of the hair is due to blocking of the follicle, whereby the sebum fails to be excreted; while the greasy masses arise from the too copious secretion of the coil glands accumulating within the upper layers of the epidermis, leading to an impairment of cornification.

The same lardaceous masses occur occasionally on the face and trunk, in the former situation especially at the sides of the nose. Those on the back have at times a salmon tint. In old people such are not uncommon, and in some wasting diseases, such as phthisis or diabetes, a similar state is encountered.

In males, fatty accumulations, allied to seborrhœa, occur under a long prepuce; in females, between and on the labia; or, in both sexes, within the meatus of the ear, as cheesy, sour-smelling, soft accretions; and in all these localities their decomposition may give rise to irritating inflammations.

Diagnosis.—In diagnosis, freedom from visible inflammatory symptoms would exclude eczema, while the scales in psoriasis are more thickly heaped and more firmly adherent. Diffused or disseminate chronic ringworm of the scalp, where the broken hairs are concealed by the healthy, does resemble and is mainly a dry form of seborrhœa. The microscope will detect the trichophyton, and the same instrument will enable a discrimination to be made from tinea versicolor of the trunk.

Treatment.—The constitutional treatment of seborrhœa consists in the administration of such tonics, usually ferruginous, as may be needed to combat the anæmia, and revivify the system, when such are called for. But local management is essential. In the first place, all accumulations on the scalp or elsewhere must be removed. A preliminary softening with oil is necessary, then washing with warm water, and either a superfatted potash soap or Hebra's spiritus saponatus kalinus, two parts of the best *green soap*, not the ordinary soft soap of the B. P., digested in one of alcohol; or a shampoo with yolk of egg, which emulsionises the fatty masses. This should be repeated at frequent, though increasing, intervals. An ointment of 15 grs. of precipitated sulphur, 10 grs. of salicylic acid, 5 drms. of lanoline, and 1½ drms. each of almond oil and distilled water, is to be rubbed carefully into the scalp at the roots of the hair once a day. Another useful pomade for the purpose of curing the tendency consists of 1 drm. of tannic acid, 24 grs. each of white wax and lanoline, and half an ounce each of almond oil and distilled water. By the employment of such measures the tendency to the perversion is arrested, and if taken in time the fall of the hair ceases. A relapse is to be obviated by careful attention to the hygiene of the scalp, by washing the head at regular intervals with 2 drms. of fluid extract of quillaia in half a gallon of warm water, and by the daily application to the hair of an antiseptic oil, made by adding a drachm of oil of eucalyptus to 2 oz. of almond oil. When the hair is too oily, the following lotion is very efficacious in lessening this, and in keeping the scalp healthy:—

℞

Chloral hyd., Ac. tannici, Resorcini, Ac. tartarici . .	āā	ʒss
Ol. ricini		♏ xv
Sp. vini rect.	ad	℥vi

Sig.—The lotion to be rubbed in once a day to the roots of the hair.

When seborrhœic masses occur on the face or body, a bland superfatted soap, such as "basmol," is to be employed for washing, and the sulphur and salicylic acid ointment mentioned above, rubbed well in, but sparingly, or 15 grs. of resorcin may replace the sulphur. The fatty accretions on the genitals and in the meatus need bathing or syringing with a boric acid lotion, and the subsequent application of an ointment of cold cream, containing 10 grs. of salicylic acid in the ounce.

The oily seborrhœa of the face generally demands systematic tonic treatment, with the use locally of a lotion containing 2 grs. of perchloride of mercury in 8 oz. of almond emulsion.

ASTEATOSIS.

An unnatural dryness of the skin, which may be either idiopathic or acquired, general or partial.

Xerodermic skins, the mildest form of ichthyosis, are not uncommon, in which the integument is harsh; and in advanced age, partly from the atrophy and anæmia associated with it, partly from diminution of perspiration, the skin manifests undue aridity. As an acquired condition, preternatural dryness, usually only affecting certain regions, may be attributed to cold winds, to the use of alkaline soaps, or contact with alkaline leys, which rob the surface of its oil, or in some trades where lime, sulphur, paraffin, benzine, or carbolic acid are employed.

Baths containing bran or gelatin, and the use of the best kinds of superfatted soaps for ablution, conduce to mollify the senile skin. This may be aided by the cautious employment of pilocarpine, which, though a direct sudorific and not affecting the sebaceous glands, undoubtedly serves to soften the skin. Lanoline, vaseline, and glycerin of starch diluted with an equal amount of neutral glycerin, are all local emollients. The dryness occasioned by occupation is difficult to treat unless the culpable agent be eschewed for a time, while the health, if impaired, is restored.

CUTANEOUS CYSTS.

These are not all connected with the sebaceous glands, nor with the hair follicle, yet it will save repetition if a general description is given, including the more common. In the first place, any *occlusion of the hair follicle above its neck* will cause an accumulation of the secretion of the sebaceous gland, distension of the duct, and the formation of a cyst; the gland itself at the same time hypertrophying. This, as Unna has shown, is most frequently occasioned by an overgrowth of horny cells, a hyperkeratosis; and according as this acts most powerfully on the hair follicle, or on the sebaceous gland, will the contents be chiefly horny or sebaceous. The old idea that sebaceous cysts arose from an opening out of the gland structure, so that the whole wall secreted sebum, must be abandoned. It is the neck which dilates, and into this the still active gland pours its secretion. Some of these cysts have an opening from which yellowish contents can be expressed without much difficulty. Such, when forced out, have often a rancid odour. Others have no visible aperture, but are smooth, rounded, or flattened bodies.

Another variety of cutaneous cysts is that known as *Milium*, an apt designation. This is a millet-seed sized, yellow or white, hardish body, seated in the skin, just beneath the epidermis. They are principally found where the skin is thin, with little subcutaneous fat, as on or in the neighbourhood of the eyelids, the cheeks, temples, and genital organs. They are, perhaps, most common in women, though met with also in children, in whom they are more truly sebaceous. The view of Virchow as to their causation is the current one, that they consist of an agglomeration of horny cells in the deeper part of the lanugo hair follicle. It is, as Unna describes it, a relatively superficial follicular horny cyst. They give rise to no subjective sensations.

But cysts, in some respects similar, may arise in another way. Such are *traumatic epidermic cysts.* In persons who are much exposed to blows or pricks with sharp instruments, round, indolent, movable tumours, about the size of a pea, appear on the sides of the hand or fingers. The origin of these has been explained as owing to the transference into the wound of a portion of epidermic tissue, capable of reproduction, which becomes encapsulated, a mode resembling that in which so-called *dermoid cysts* are produced. These arise, as Unna expresses it, "in an early embryonic period, by the wandering of part of the epidermis into the subcutaneous tissue." These latter include, as shown by Török, the *atheromata* or *wens*, so often seen on the scalp. The contents are various, but all referable to the epidermis. Thus they may be soft and pasty white, or brownish and thin, or like honey, or glistening from the presence of cholesterin. Some are in the substance of the skin, as are the common *chalazion cysts* of the eyelid; others, as the wen, are distinctly subcutaneous. Their capsule consists of connective tissue differing in thickness.

Such cysts may rupture, and from the surface so exposed a *cutaneous horn*, corresponding to the hair altered in character, may sprout, or a cauliflower-like mass, not very dissimilar to an exuberant epithelial cancer, may develop, as in a case described by Hutchinson, where the greater part of the scalp became so transformed.

In the treatment of cysts, those which exhibit a visible opening may need nothing more than periodical expression of their contents. The cyst gradually becomes smaller, and eventually disappears. The closed cysts must be dissected out, under carefully instituted antiseptic precautions. Those on the eyelids should be excised from the conjunctival aspect under cocaine, to obviate leaving a noticeable scar. In the case of milium, the thin covering layer should be scratched through with a fine pointed knife, then the shotty body can be readily turned out.

COMEDONES.

Black spots or puncta, which have their seats in the conjoined duct of the hair follicles and sebaceous glands, seen on the face, back, and chest. They are open cysts containing horny material and sebum.

Etiology.—The cause of comedones seems to be an undue thickness of the horny layer, with a want of tone in the muscles of the skin, especially the arrectores pilorum. In those who work among petroleum or tar, the stimulation which these substances exert on the corneous layer promotes their production, by inducing closure of the follicular mouth. Some oily seborrhœa often coexists, as if there is a compensatory over-action on the part of other glands. Double comedones arise, according to v. During and Török, from a fusing together of two or more sebaceous gland ducts, by intradermal suppuration, the secreting part remaining productive. The *Acarus folliculorum*, found head downwards in some comedones, seems, at least in man, a harmless concomitant.

Symptoms.—These are more common in dark than in fair-complexioned persons. The skin itself is apt to be inactive, muddy, and deficient in transparency. The anæmia, also so frequently coexistent, is partly real, partly apparent, the vascular plexuses being veiled by the non-translucent skin. Met with occasionally at all ages, they give most trouble between puberty and 30. In those subject to them, though never entirely absent, they are not at all times equally in evidence. The sluggishness of the skin associated with them is influenced by inactivity of digestion or irregularities of menstruation. They are generally dotted without arrangement over the areas mentioned, but may be grouped symmetrically on the flush regions of the cheeks, and are then smaller than ordinary. When pressure is exerted, a worm-like cylinder escapes, consisting at its upper portion of horny material, darkened, according to Unna, by retention, while the deeper is tallowy and soft. Sometimes comedones occur as doublets or triplets, as first described by Ohmann Dumesnil. These are most often seen on the back, and in those affected with acne. The contents, lemonade-bottle-shaped, expressed from these are blackened at both extremities.

Treatment.—The treatment of comedones is not always rapidly satisfactory. It is necessary to dislodge them, not, however, attempting to press them entirely out. For this purpose a watch-key, or one of the many extractors which have been invented, may be used. For washing, either green soap and warm water, or sometimes better, a resorcin and salicylic acid soap, which tends to thin the corneous layer, may be employed. If not very large or numerous, the following paste, recommended by Unna, should be applied at night, with the eyes closed, to prevent recurrence:—
℞ Kaolini, semi unciam; glycerini, drachmas tres; aceti, drachmas duas. The ointment of sulphur and salicylic acid, described under Seborrhœa, is a stronger application; or Zeissl's paste, consisting of equal parts of precipitated sulphur, glycerin, rectified spirit, carbonate of potass, and ether. These should be applied at night, carefully avoiding any excess of stimulation. The parts are to be washed with a bland soap in the morning, and dusted with emol keleet, or 10 per cent. of boric acid in talc. At the same time the general health needs suitable attention.

ACNE VULGARIS.

Inflammation, either mostly confined to the pilo-sebaceous follicle or producing also suppuration in the sebaceous gland, usually preceded by the formation of a comedo, and limited in locality to the face, shoulders, and chest, in time to the period between puberty and maturity. The connection between the comedo and acne is so close, that though the latter properly belongs to the class of inflammations, it is better to consider it here. It constitutes one of the commonest diseases of the skin, and if of any intensity is apt to leave, if untreated, indelible marks, in the form of pitted scars.

Etiology.—The causes of acne are chiefly local, but there are predisposing influences at work. The main agency in operation is that vascular pressure, which is inseparable from active functional development, as of the transformation of the downy investment of the cheeks of the boy, to the strong, thick, and deeply implanted hairs of the whiskers and beard of the man. This nisus exists in the female, yet rarely receives expression, save in a few young women, whom the unnatural growth of hair asexes. There is a restraining influence in women, which loses a portion of its power towards the wane of sexual life, and then the change of lanugo into coarser hair partially or even extensively occurs. The less regularly, steadily, and perfectly does the hairy transformation go on in youths, the more tardy the evolution of the manly beard, the more intense, other things being equal, the tendency to the development of acne. That sluggish condition of skin which favours and promotes the superficial hyperkeratosis of the epidermis, which is the first stage of acne, is a feature of such cases. Suppuration has hitherto been believed to be dependent on the incursion of ordinary pyogenic organisms, but Unna holds that this is erroneous, since they were absent in a series of cases examined by him. He found in all instances of true acne a special bacillus, having an irregular arrangement within the comedo. It is small and plump, according to him "fat-loving and fat-consuming," and it is the efficient agent in producing the suppuration. This suppuration may be shallow (impetiginous), or deep (follicular). Schütz from his observations concluded that Unna's acne bacillus was merely the *Bacillus coli communis;* but Sabouraud identifies the bacillus which produces what may be termed the seborrhœic cocoon—the comedo, in short—with that in acne, thus bringing these two diseases into still closer connection. In some persons horny and sebaceous cysts arise, deep in the skin, or having their seat in the subcutaneous tissue. These may suppurate, and hence it comes that when one of these deep oval or rounded elastic-feeling structures is punctured, we sometimes evacuate a thick oily-looking fluid, sometimes pus. While these are shortly the local causes of acne, various functional disturbances of the sexual and digestive systems are distinctly operative in producing temporary aggravation. Payne has seen cases where fresh outbursts invariably occurred if salt liquors, sweets, or rich food were partaken of. These act by reducing the nervous tone in the parts affected, and so diminish the restraint on the proliferation of implanted organisms, which is exerted in a healthy state of tissue.

Symptoms.—The earliest stage of acne is a degree of superficial hyperkeratosis of the epidermis, which, advancing to the follicular openings, closes them, and thus leads to the production of comedones. These are more or less prominent in individual instances. The appearances in acne vary much with the soil, and so do the number of lesions. The florid and clear complexioned have few, though perhaps rather large and angry spots; the torpid, sluggish, and anæmic have many, which may be deep-seated, widely diffused, and obstinate. Hence the ultimate effects are different. The scars, if any remain at all, are in the former few and insignificant, but in the latter they are often numerous and disfiguring. The acne pustule has no determinate shape or colour. It may be bright red, acuminate, with more or less areola, or even with a hard crimson base, or it may be elongated and violaceous, nodular or subcutaneous. The skin looks thick, there is some oily seborrhœa, but the subjective sensations are not pronounced. Heat, perhaps irritability, seldom itchiness is complained of.

Diagnosis.—This is seldom difficult, though there are forms of perifolliculitis which simulate it pretty closely. The age limits, which are only occasionally exceeded, the localisation, the coexistence of comedones, and the almost entire absence of subjective symptoms, are usually sufficient for its identification. The small pustular syphilide, which in some respects resembles it, is uniformly and widely distributed; there are no comedones, and as it appears within the first year after infection, there are constitutional symptoms to be discovered, if carefully enough sought for. The folliculitis occasioned by contact with tar or paraffin no doubt has an affinity with acne, but may occur at any age, and is directly traceable to the irritation caused by these substances. A pustular eruption in persons beyond the usual limits in age, should suggest inquiry as to whether bromides or iodides have been taken. The differentiation from rosacea will be found under the latter.

Treatment.—In treatment, local measures should be primarily considered as being the more important, though constitutional regulation is, in most cases, essential as an adjuvant. In the first place, something must be done to neutralise the hyperkeratosis, which has led to the closure of the sebaceous gland-ducts. The natural greasiness of the skin in acne commonly permits of, or at least does not contra-indicate, the use of soap. That selected should, however, be either a good neutral or superfatted one, preferably, also, one of which the alkali is potash rather than soda. Those substances which hasten desquamation act favourably on the hyperkeratosis; thus, superfatted soaps, containing a little sulphur, or resorcin and salicylic acid, used cautiously, may prove advantageous. Unna has shown that in the process of healing, in the slightest cases, only the comedo, with its bacilli, are thrown off in the suppurative process. Thus it is good practice to endeavour to prevent the formation of a pustule by judiciously pressing out the comedones. The reactive effect of douching, spraying, or bathing with very hot water, is sometimes useful, employed after the pressure, and not continued too long. Much good may often be accomplished in a short space of time by scraping the surface with the dermal curette, or Volkmann's sharp spoon. These measures must be combined with the use of applications which, while acting more slowly, have a more continuous effect. In general, lotions are more pleasant and efficacious than ointments. The chief ingredient in these is commonly sulphur, which is of value in two ways. One, by causing a slight but persistently increased exfoliation of the outer epidermic layers, in which the pilo-sebaceous gland-duct participates sympathetically; another, by directly annulling the vitality of the pyogenic organisms, whatever their nature. Perhaps the most generally useful is the well-known Vlemingkx solution, somewhat diluted—℞ Sulphuris sublimati, drachmas duas; calcis vivæ, drachmam unam; aquæ destillatæ, uncias decem. The quicklime and the water should be first mixed, the sulphur added, and the combination boiled slowly down to 6 oz. in a porcelain capsule. This is to be put on thinly at night with a camel's-hair pencil, and allowed to dry. If too strong, as is shown by the production of redness and dryness of the skin, the lotion should be diluted; or, conversely, it may sometimes be employed stronger to resistant skins. If an ointment be preferred, that suggested by Boeck of Christiania, is often prompt—℞ Acidi salicylici, camphoræ, āā 0·30 to 0·50; saponis viridis, 1·0; zinci oxidi, 2·0; sulphuris præcipitati, 10·0; olei cetacei purificati, 12·0. The odour of the whale oil is masked to a large extent by the camphor, while its penetrating power is greater than any other excipient.

Since all the measures recommended are more or less irritating, they are best employed at night. In the morning, the lotion should be washed off with plain tepid water, the ointment with superfatted soap and warm water, and then a soothing dusting powder is to be applied. For this purpose emol keleet suits well, and adheres excellently, or 10 per cent. of boric acid in powdered talc. Early pustules may be caused to abort by touching each with carbolic acid or lysol, and varnishing with flexible collodion or krystalline. If they are large and angry, it is better to cover them with a piece of Unna's carbolic and mercury plaster muslin, which may be changed every second or third day. The deep cysts may require incision and curetting their inner surface, but they often disappear if the sulphur treatment is persevered in.

Extensive and chronic cases of acne sometimes demand another method. This consists in the application of Unna's salicylic and creasote plaster muslin, using either the 15–30 or the 20–40 strength. Strips are closely applied to the affected parts for from three or four days to a week, till the surface is rendered pretty raw, the plaster being renewed and the skin carefully cleansed every twelve hours. The treatment should be continued till all apparent pustules have flattened down, and the integument shows of a red colour, with deeper-hued points, the seat of the pustules. The plaster is then laid aside, and the skin is dressed with Unna's zinc ichthyol salve muslin, till the epidermis is restored. The cycle, in some cases or parts, may need repetition, but eventually such amelioration is produced, that the lotions or ointments indicated may be resorted to.

In directing internal treatment, all discoverable errors must be corrected. Thus the management is frequently more hygienic than medicinal. Aperients, hæmatinics, tonics, and gastric and intestinal correctives are indicated in different instances. The diet needs regulation, often modification. The state of the sexual functions requires attentive consideration. In many cases exercise suited to the person must be enjoined. Riding, cycling, golf, and particularly swimming in the sea, or in well-constructed baths, are advantageous; the maintenance of the due activity of the skin in some way is essential. Acne is curable, but only by judicious perseverance in well-chosen treatment. After all active phenomena have been banished, gaping follicles may still stud the countenance; these may be rendered less conspicuous by electrolysing each, as in the removal of hairs.

ROSACEA.

A chronic disease of the face, limited almost entirely to the nose, cheeks, forehead, and chin, characterised by hyperæmia, dilatation of the venules, nodulation, or pustulation, and, in severe cases, hypertrophy.

Etiology.—A fertile cause of rosacea is chronic indigestion; in men this is perhaps more often gastric, in women intestinal, but in both the disturbance is chiefly catarrhal. This is responsible for many red noses, so disfiguring, and the source of so much ill-natured comment. Occasionally such are due to chronic hypertrophy of the nasal mucous membrane, especially that over the middle turbinated bone. This gastro-intestinal catarrh may be traced to the abuse of tea, or of alcohol, but is also one of the consequences of a sedentary life, under bad hygienic conditions. Some of the worst cases, of the highest degree, have been encountered in persons not given to spirituous liquors. Though rosacea is met with at all ages in women, yet it is most prevalent at the close of menstrual activity. Menorrhagia is not infrequently associated with it. Exposure to excessive heat, or over-exertion in such heat, has seemed sometimes to start it. Seborrhœa of the scalp may coexist, but something more is needed to originate the disease. This consists in some remotely-acting disturbing agency on the notoriously sensitive vascularisation of the face. Alcohol has such an effect, and the ailment is usually worse before a menstrual period, or is aggravated by successive pregnancies.

Symptoms.—Sometimes the disease is confined entirely to the nose, the tip of which is red, swollen a trifle, and glossy. This approximates to chilblain, but is more persistent. Or there may be an erythema, taking the type, as Besnier describes it, of a flush, at first intermittent, then remittent, and finally permanent, with exacerbations. This is common in women, even in girls. Most frequent of all is the seborrhœic, which Unna classes as one variety of his seborrhœic catarrh, and originating from transference from the scalp. There is redness, either of a bright crimson or a dull purplish hue, more or less diffused over the areas above named. The colour varies according to the activity or the reverse of the peripheral circulation of the individual, the degree of thickness of the epidermis, or the temperature of the air at the time. If the skin is thick, the surface may be greasy; if thin, there is slight scaliness, and the aspect reminds one of an erythematous dry eczema. On inspection, there are discoverable some dilated and tortuous venous radicles, or these may be numerous and large. Unpleasant sensations of burning are often complained of, particularly on suddenly entering a hotter or cooler atmosphere, or after the ingestion of hot fluids, such as tea. The erythema may become deep, so that a swollen condition of skin is produced, yet it is usually cool to touch.

This constitutes the first grade, but on this abnormal basis papules and pustules arise. These are sometimes superficial and small, sometimes hard and deep, so that the complaint may in one resemble a pustular eczema, in another a highly inflammatory acne. No comedones are visible, however, and the furuncles own an ordinary pyogenic origin.

The third grade is marked by definite hypertrophy. This may be purely fibrous, the new tissue being formed at the expense of the normal spindle cells. Or there is an increase in the size of the sebaceous glands themselves, studding the surface like bits of orange-peel. Ordinarily the process is a combined one, though this extreme development is seldom seen in women, and even in men is rare. There is great coarseness and greasiness of skin, much connective tissue hypertrophy, so as to give rise to irregular overgrown masses, which hang from the nose or appear as excrescences on the cheeks and forehead, simulating in the latter situations the leontiasis of leprosy.

Diagnosis and prognosis.—Rosacea may be simulated by syphilis, but in the early stage the concurrent general symptoms, in the later the crescentic arrangement of the elements, the ulceration, or scars left, will serve to distinguish. In alcoholics, however, the tuberculous syphilide of the centre of the face can sometimes only be differentiated from rosacea with nodulation by the therapeutic test. A good illustration of this will be found in Wilson's "Portraits," Plates A, B. Some forms of lupus erythematosus likewise resemble rosacea, The face must be scanned for scars; if any, though faint, are found, it is not rosacea. The disc-like arrangement, the splintery scales, with tags passing into the follicular openings, the implication of the lobe of the ear, and the absence of venous telangiectases, are typical of the erythematous lupus. It is separated from acne vulgaris by the age of the patient and the non-existence of comedones.

The prognosis in rosacea of the first or second grade is usually good, and the same may be averred of the third, provided it is restricted to the nose, or merely occurs as isolated bossy growths on the cheeks or forehead. The most obstinate forms are that limited to the tip of the nose, apparently the most trivial, and the rare diffused hypertrophic variety. Even though dispelled, it may recur, and such relapses yield less readily than the original trouble.

Treatment.—Constitutional treatment is more necessary in rosacea than in acne. The diet, in particular, must be simple and non-stimulating. Every article which tends to cause flushing must be rigorously eschewed. Thus, hot liquids of all kinds, curries, condiments, and prepared sauces, are inadmissible. The amount of butcher's meat should be limited, and replaced largely by white meats and fish. Green vegetables, certainly, plain salads, free from dressing, are usually permissible. Fruit, if ripe, seldom does harm. Water should be the sole beverage; milk may be partaken of freely, best with the addition of lime water, but is to be regarded in the light of a food. Milk puddings, and farinaceous articles in general, suit most cases.

The hygiene of the skin, as a whole, requires attention. Warm baths, especially sulphur, act beneficially, but Turkish baths are contra-indicated. Bearing in mind the implied etiological connection with seborrhœa, the scalp ought in all cases to be examined for this, and if found affected, treated.

The local treatment must be chosen in accordance with the severity of the case, and the degree of resistance of the skin. Sulphur is one of the most efficacious remedies, but its action needs watching. One drachm of precipitated sulphur, in 4 oz. of calamine lotion, forms a suitable combination. A little of this is painted on at night, washed off without soap in the morning, and the face dusted with emol keleet, or boric talc powder, as in acne. Sometimes the diluted Vlemingkx solution may be used in those with more greasy skins. The dilated venules require scarification with a fine iris knife, followed by bathing with warm water, or, if large and tortuous, they may be destroyed by electrolysis. The hypertrophic masses can be shaved off with a sharp razor. The nose can thus be trimmed to an appropriate shape. Bleeding is free, but easily checked.

The strictly medicinal treatment comes last in importance. Ichthyol perhaps gives the best results. It should be administered in doses of 3 to 5 minims, freely diluted, two or three times a day. It should be taken when the stomach is empty. Schütz advises that remedies which arrest fermentative changes should be given in the interval between meals. Three to 10 minims of the sp. thymol. may be taken at 10 A.M. and 5 P.M., in a glass of water. Small doses of hydrochloric acid, in free dilution, with meals, also do good.

ADENOMA SEBACEUM.

Firm whitish or yellowish, sago-grain-like, solid flattish papules, over which, in some cases, minute blood vessels ramify, and consisting of hypertrophied sebaceous glands. Rayer, Addison, and Gull described the first cases, but Balzer, who attached the name, identified it. The best account is that given by Pringle.

Etiology and symptoms.—It occurs in two forms, one the congenital. The eruption consists of indolent, firm, whitish or yellowish solid papules, or little tumours, smooth or warty, embedded in the skin at different depths, or projecting from it, and varying in size from a pin's point to a small pea. Though thickly crowded together, these lesions have all a distinct boundary line, and never appear to coalesce to produce composite patches. There is no solution of epidermis, or anything suggestive of excretory duct, but when pricked with a needle, white matter can be easily expressed from them, recognisable under the microscope as inspissated sebum. Associated with these there is a certain amount of concomitant vascular hypertrophy or telangiectasis, which, however, varies within wide limits. It may be scarcely noticeable, or is the feature which most prominently attracts attention. Those affected have been observed to be intellectually below par, and have chiefly been females. This is a summary of Pringle's account; but there is a second form, which does not develop so early in life, and may be met with in those of quite average intelligence. An instance has been recorded by myself, and another by Pollitzer. The first part of the description given above applies fairly well to both, though in Pollitzer's the colour was reddish. In neither were there any telangiectases. The disease is a rare one, and in situation seems confined to those parts of the face affected by rosacea. There are no subjective sensations. It may be symmetrical or more or less unilateral. The appearances are in miniature those of hypertrophic rosacea, and an examination into the structure of the growths bears this out. They are made up of hypertrophied, at times of hyperplastic, sebaceous gland tissue. Unna doubts if they should be considered as adenomata, or growths in a new direction. Some ought, in Walker's opinion, to be rather classed as lymphangiomata. Probably all cases are truly congenital in origin, though they do not definitely appear till later.

Diagnosis.—The disease bears the closest resemblance to epithelial cystadenoma, a condition to which nearly every observer has attached a new name. The diagnosis is indeed scarcely possible without a microscopic examination, but a comparison of the descriptions will aid.

Treatment.—This is only satisfactory if the number of elements is limited, so as to permit of the whole being excised without the production of a disfiguring scar. Electrolysis lessens the conspicuousness, and the remedies for rosacea will tend to remove the telangiectasis. In my case, considerable benefit resulted from the vigorous application of a 40 per cent. resorcin paste.

ACNE, OR FOLLICULITIS VARIOLIFORMIS.

Groups of peculiar pustules, occurring principally on the upper part of the face, but extending among the hair, which finally leave scars like those of smallpox. Hebra first described this rare eruption under a name originally used by Bazin to designate molluscum contagiosum epitheliale. The commencement consists in the formation of papules or nodules on the forehead, temples,—advancing among the hair,—or more rarely on the neck, chest, or back. In their centres is a hard point, round which a ring of pus appears, surrounded by a narrow areola. A firmly attached scab is thus produced, beneath which there is more or less necrotic softening, so that, on its removal, a pit is left, dark red or brownish, eventually becoming a white cupped scar. The ailment is extremely chronic, and Crocker believes that in some cases, at least, syphilis is a predisposing cause. Unna holds that it is caused by the growth of a special bacillus in the lanugo hair follicles. This does not give rise to a comedo, as in acne, though it exercises a stimulating influence on the follicular epithelium. The crusts are due to the development of seborrhœal organisms in the scales at the follicular mouths, and it is to their active development that the necrosis is owing.

The diagnosis from a pustular syphilide lies in the restriction of the area affected, but there may be difficulty with a recurrent tertiary pustular eruption. Treatment consists in the removal of the crusts by starch poultices containing boric acid, and applied cold. Then an ointment containing sulphur and ammoniated mercury, made with lanoline, should be rubbed in. Internally Crocker recommends iodide of potass.

THE SWEAT GLAND APPARATUS.

In the same way as in the sebaceous gland system, so in the sweat or coil gland one, we may have increase or diminution of the secretion, various forms of perversion, and organic changes connected with the apparatus itself.

HYPERIDROSIS.

Excessive secretion of perspiration, occurring under circumstances not ordinarily habitual.

Etiology.—The copious perspiration which follows on exposure to heat, as of the sun or a fire, or which results from physical exertion, is not, strictly speaking, hyperidrosis, yet there can be traced all degrees between this physiological moisture and that extreme of the pathological, where the individual can hardly make a movement, or experience an impression, without perspiring. The condition may be general or local. The facility with which perspiration is induced varies, the obese sweat more readily than the spare. A deficiency in tone is the chief factor in predisposing to morbid hyperidrosis. When there has been a disturbance for a longer or shorter time of the heat-regulating centres, when fever has existed, the instability of the centre persists after the temperature has become normal. Sudden and profuse perspirations are apt to occur, and as suddenly to cease, producing then by the evaporation a sensation of chilliness in the relaxed skin. Though there is, for the most part, a relationship between the activity of the cutaneous circulation and the secretion of sweat, still this is not constant, and the skin may be congested yet dry, or pale and drenched with perspiration.

Pathology.—Pathologically there is a disturbance of vasomotor control, for the side of the head affected with migraine can be seen perspiring freely, while the other is dry. The profuse sweats which occur during slow somatic death, in such disorders as pneumonia or pyæmia, are connected with a vasomotor paralysis consequent on poisoning of the central nervous system.

Symptoms.—Local hyperidrosis may affect those parts where the sweat glands are particularly large, but where sebaceous ones are likewise present, as in the axilla or on the perineum or it may be confined to localities where there are sudoriparous glands alone, as the palms and soles, or, as in rachitic children, the head only may perspire too freely. Of these the axillary is the one most closely connected with psychical reasons. Sweat may be seen to pour from the armpits of patients stripped before an audience of students. If the palms suffer, the hands feel cold, damp, and clammy, contact is unpleasant; the skin becomes sodden, tender, sometimes has a delicate, pinkish hue. Gloves, if worn, are rapidly soiled, and no fine work can be engaged in, since, if touched, it acquires a greasy stain. Persons so affected are nearly always in a lowered state of health, are more often females than males, may be hysterical or neurotic; some mental strain or shock is occasionally blamed. This moist condition of the hands is peculiarly apt to be found in such as are addicted to chronic alcoholic excess. In these the skin is flabby, porous, fatty, and with a degree of velvety softness. Axillary hyperidrosis is promoted by wearing impervious "dress preservers." The abuse of tea tends to produce in some cold perspiration of the hands and feet.

Treatment.—In the treatment of general hyperidrosis we must have resort to a tonic medication. That which succeeds feverish states commonly ceases gradually with progressive convalescence, but may be expedited by the administration of quinine, the mineral acids, and of tinctura agarici or agaricin, guarded, if necessary, by the addition of minute does of nepenthe or Dover's powder. The local forms require similarly suitable roborant measures. The system should be braced by cold or tepid salt shower or plunge baths. Free application of the following powder to the parts is most effective: ℞ Acidi salicylici, 3·0; pulveris amyli, 10·0; talci, 87·0. Lotions of permanganate of potass are often useful.

BROMIDROSIS.

A functional disorder of the sweat glands, in which there is an offensive odour, combined with more or less sweating.

Etiology.—Thin has found in these a profusion of micro-organismal growth, his *Bacterium fœtidum*. This may either be the active ferment, or finds a congenial soil in the putrescent perspiration. The complaint is frequently met with in female domestic servants, though not confined to them or to one sex. It is encountered in connection with anæmia, and is apt to be worse during menstruation. Sexual excitement also occasions degrees of bromidrosis.

Symptoms.—There is not necessarily an excess of perspiration. Sometimes a heavy disgusting smell exhales from the whole surface of the body, intensified should perspiration be increased. This may resemble that of garlic, or may be merely strong. In association with some of the exanthemata, particularly smallpox, the emanations are peculiarly disagreeable; the odour in typhus is of a special nature, and the penetrating acid sweat in rheumatic fever is well known. When certain drugs, as sulphur, iodine, asafœtida, or musk, are taken, they manifest their presence in the system through the skin. Much, though not all, of the close atmosphere of ill-ventilated bedrooms after a night's occupancy is due to bromidrosis.

This rather proceeds from the feet or axillæ or perineum than from the general surface. Bromidrosis pedum is the most common. The soles are usually sodden, with a broad, pinkish margin. There is sometimes more or less flat-foot coexisting; the feet are tender, moist, and clammy; walking is painful, and in severe and neglected cases the skin inflames and peels. The odour is partly due to the rapid putrefaction of the sweat and the decay of the macerated epidermis, but more particularly to the fact that the coverings of the feet, the stockings and shoes, soak up this unhealthy perspiration, and thus the decomposition and the evolution of rancid odours proceed more quickly. Though many such cases can be permanently cured, still there is a considerable tendency to recurrence in some.

Treatment.—The treatment consists in correcting any discoverable constitutional fault. The most sedulous cleanliness must be enjoined as an essential to cure. This must not only embrace the necessary care as to the skin, but specially frequent changes of stockings, shoes, and underclothing. The stockings should, if possible, be boiled, or plunged in boiling water. The feet are to be washed with soap and warm water every night, dried, then powdered freely with the salicylic powder mentioned under hyperidrosis. In the morning, before the stockings are put on, the powder should again be applied. In washing the feet the use of a loofah is advantageous, as serving to remove the masses of epidermis.

CHROMIDROSIS.

In investigating any alleged instances of coloured perspiration, two sources of error must be eliminated. One is simulation, since this is one of the ways in which the hysterical temperament may manifest itself; the other, that contact, in pursuance of occupation, with various tinctorial substances may indirectly tinge the perspiration. Still a sufficient number of well-authenticated instances have occurred where the sweat has assumed a blue hue, or a red one, to permit of this being recognised. As to blue sweat, described by Parrot, little definite is known. Red perspiration is encountered chiefly in association with hyperidrosis; it affects the axilla, more rarely the pubes, and those who have red or very fair hair. The linen or woollen material worn next the skin is stained red in these regions. Axel Key was one of the first to notice that the hairs were encrusted by a peculiar fungus, which has been found to resemble the *B. prodigiosus*, and Balzer has found similar zooglœa in epidermic scales scraped from the parts. The microbes attach themselves to the epidermis of the hair, especially to places where this is eroded or defective, forming yellowish or glutinous masses, not unlike the ova of the pediculus on cursory observation. Sometimes there is slight pruritus and dermal irritation. The source of the complaint has so far eluded observation. *Hæmatidrosis* is a purely neuropathic phenomenon; it is a diapedesis of red blood corpuscles emanating from the sudatory vascular network, occasioned by perverted nerve influence, but it is not a true skin disease.

Though in cases where the urinary function is defective the aid of the skin is often called in, this is rather as a hydragogue, a derivative, or a revulsive, than as an alternative excretory organ. Hence *uridrosis* can scarcely be seriously spoken about. When urea has been substantiated in the perspiration, it has been chiefly in that secreted in the last stages of some mortal disease, and it cannot be looked on as a constituent. Much the same may be said of *galactidrosis*, or milky sweat.

The treatment of red sweating consists in shaving the parts and applying ether, as this agent has at least produced a temporary cessation.

There are certain morbid conditions closely associated with hyperidrosis, yet which are not, in the opinion of competent observers, directly traceable to the sweat glands or their ducts. One of these is

DYSIDROSIS OR CHEIROPOMPHOLYX.

An eruption of deep-seated, sago-grain-like vesicles on the hands, less often on the feet, in persons who perspire readily, tending to recur in warm weather. This disease, not a very common one, was described by Tilbury Fox under the first term, by Hutchinson under the second; and its morbid anatomy has been investigated by Fox, Robinson, Santi, and Williams.

Etiology.—It is most frequently seen in young women, chiefly those of neurotic temperament, who have become lowered in health from worry or overstrain. The hands are generally too moist, and anæmia may coexist.

Symptoms.—It commences with sensations of burning or tingling, along with the appearance of vesicles in the interdigital spaces, and by the sides of the fingers, on the palms, or even over the whole hand or foot. These seem deep, are isolated or in groups, and if the latter, may coalesce so as to form large and prominent bullæ. Particularly on the palm they resemble boiled sago grains implanted in the integument. Fox noticed that in the earliest stage small transparent rings of fluid are visible round the sweat pores, but with increase in size this disappears. The contents of the vesicles are neutral or alkaline, subsequently turbid. When fully developed there is some swelling but not much redness, though on the back of the hand the advancing edge is vesicular, the included area pink and tender. Except at the sides of the fingers the vesicles seldom rupture spontaneously, but dry up into a hardish crust or scale, which slowly separates in a couple of weeks. The reddened skin beneath is not prone to ooze.

The vesicles lie in the rete immediately below the basal horny layer, but it is disputed whether there is a direct connection with the sweat pores or not. Unna admits they are in close approximation, but considers them inflammatory. He attributes the disease to the action of a special bacillus, dormant under ordinary circumstances, but roused into proliferation by the sweating in summer, or induced by lowering ailments. This is naturally most active in the neighbourhood of the sweat pores, the watery portion of the perspiration being drawn, according to Unna's view, from the juicy tissues round the duct canal.

Diagnosis and treatment.—The diagnosis from eczema and scabies lies in the limitation to the hands and feet, the fact that the vesicles dry up in place of rupturing, the spontaneous recovery, and the liability to recur in summer. The management consists in promoting this tendency to dry, by the use of antiseptic and desiccating powders, such as 3 per cent. of salicylic acid in talc, or 10 per cent. of boric acid in the same excipient, to which may be added 10 per cent. of powdered starch, which causes it to adhere better should the vesicles break. If the itching and tension are marked, a useful preliminary is the envelopment of the affected member in a thick layer of cold boric starch poultice, sheathed on the side next the skin by a layer of muslin. Mineral acids and a bitter or some acid ferruginous preparation may be administered internally.

MILIARIA AND SUDAMINA.

Pin-point to millet-seed sized, thickly crowded, and widely diffused inflammatory papules or vesicles, attended by pricking or burning sensations, are seen in connection with excessive sweating, and are evoked by heat.

Under this heading several allied ailments are included. One of these is the complaint, so troublesome to residents in hot climates, called *prickly heat.* In it numerous minute, acuminate, bright red papules make their appearance quite suddenly over great part of the body, their eruption being preceded or accompanied by more or less perspiration. The irritating tingling experienced coincidently, gives origin to the name. This is probably due to sudden distension of the blood vessels, exercising pressure on the cutaneous nerve terminals. Vesicles are met with along with the papules, but are the sole manifestation in *sudamina*. When there is high temperature of the body, especially if too warm a regimen is pursued, in the course of one of the eruptive fevers, or of an acute inflammation, as pneumonia, there is sometimes observed a copious outburst of small, clear, discrete vesicles. At first they may closely resemble drops of dew standing on the surface. At other times their contents are milky, or round each is a narrow bright red areola, so that they appear as white pearls on a crimson groundwork. They are not in any sense critical, and have no pathognomonic significance. Much the same is the *red gum* or *strophulus* of infants, a closely set acuminate papular or vesicular rash, evoked by wearing thick flannel binders or undergarments, or from the child being held too continuously to its mother's side.

Török has shown that the complaint is an inflammatory one, but with a direct relation to the sweat ducts. The vesicles begin in the deepest part of the horny layer, but extend into the prickle layer, forming single-chambered vesicles. Though in itself distinct from eczema, yet it may form, according to Unna, the starting-point of a seborrhœic dermatitis.

Treatment is directed to reducing the temperature by lessening as far as is judicious the coverings of the body, and by substituting as far as possible linen or silk for woollen materials. Too much washing should be avoided; dusting powder, such as emol keleet, useful for its absorbent and antipruritic properties, or boric acid with talc, may be employed. A lotion recommended by Boeck is valuable; this is made up of 100 parts each of talc, pulvis amyli, lotio plumbi subacetatis dil., and 1 per cent. solution of boric acid in water, with forty parts of glycerin. This when used is further diluted with twice as much cold water, and dabbed or painted on. In prickly heat absolute rest in a cool well-ventilated room, with light clothing, unstimulating food, and the use of such acidulated drink as lemon squash or lemonade, combined with the local applications mentioned, give speediest relief. Inunction with vaseline or bland oil after the morning bath is useful as a prophylactic, in prickly heat.

HIDROCYSTOMA.

Cysts of the sweat gland duct, characterised by the appearance of firm, translucent, pin-head to split-pea-sized vesicles, on the upper part of the face, associated with hyperidrosis. Robinson of New York originally in 1884 described the condition; since then a number of instances have been placed on record. One of the most recent accounts is that of Thibierge.

Etiology.—Nearly all the examples have occurred in women, either in middle life or slightly beyond it, though in them the complaint had existed for many years. They had been employed in general housework, or in cooking or washing, labouring especially in a warm, often moist atmosphere, which caused them to sweat profusely. Robinson has shown, and his observations have been in the main confirmed, that in the sweat coils, which are partially dilated, there is a granular secretion. Above this, in the deeper part of the corium, the duct is dilated and becomes a cavity, lined with flat epithelia, and containing a clear fluid. The condition is one closely connected with excessive sweating, but the cause of the vesicular formation has not, so far, been satisfactorily accounted for.

Symptoms.—The eruption is invariably worse in summer, and becomes better, if it does not entirely disappear, in winter. The recurrence of the menstrual period and emotional states seem in most cases to aggravate it. In situation it is confined to the upper part of the face and forehead. It is usually bilateral, but not always symmetrical in distribution. The elements consist of clear, tense, shining, hemispherical vesicles, varying in size from a pin-head to a large pellet. They are at first at least deep-seated, and resemble when small a boiled sago grain. In some a bluish tint can be discerned, deep in their floor. A few capillaries may ramify over them. There are no concomitant signs of inflammation, and though itchiness is occasionally experienced when they develop afresh, as a rule, there are no marked subjective sensations. The contents are limpid and acid in reaction, the vesicles themselves persist for a considerable time; eventually, most dry up and scale off, leaving a faint pigmentary stain. Cheiropompholyx has not been met in association with them.

The **treatment** is similar to that recommended for miliaria.

POROKERATOSIS.

This is a warty overgrowth affecting mainly the sudoriparous ducts, localised principally on the backs of the hands and feet, but involving also the extremities and head. The disease has only recently been described by Mibelli and Respighi; the illustrations provided by the former in Part IX. of the "International Atlas of Rare Skin Diseases," convey an accurate impression of its features.

The disease commences by the formation of small horny discs, which extend in area, and multiply in number. When fully developed, they are raised and flattened in the centre; there is a rough, horny epidermis, studded with minute acuminated projections, limited by a narrow, wavy, almost prickly margin. They are yellowish brown in colour, and vary in size from a lentil to a sixpence. On the forearm, the sinuous, hard, well-defined margin forms an extensive line. Beyond the disfigurement, there do not seem to be any annoying symptoms, and though some patches spontaneously fade, the complaint remains for years little altered.

The epithelial overgrowth affects mainly the sweat pores, which become filled with black horny masses. At the same time there is a dilatation of the vessels of the papillæ, with cellular infiltration. The marginal wall consists of thickened horny layer. One might suspect a parasitic cause, since, in Mibelli's case, other members of the family were subsequently attacked. Ducrey and Respighi, however, think that it is not contagious but hereditary. Unna regards it as chronic inflammatory in nature. Hutchins has observed a case in America. It may be well to bear in mind the tendency which arsenic has, as Pringle has demonstrated, when long taken, to produce corn-like growths on the palms and soles, combined with hyperidrosis.

THE HAIR SYSTEM.

HYPERTRICHOSIS.

An unusual development of hair, either on regions where it is not ordinarily found, or only as down.

Etiology.—The cause of this abnormal vigour in the development of hair is often obscure. It occurs in some young women who present a masculine type; in others, it is connected more or less distinctly with ovarian irritation, deficient or irregular menstruation, or other unhealthy states of the female generative organs. It is so commonly observed at and after the menopause, that one must ascribe an inhibitory influence to the ovaries, during their period of activity, on the abnormal development of hair, as one of their functions.

Symptoms.—The condition, regarded as a disease, is almost limited to that met with in women on the face, neck, or bosom, where the nearly imperceptible down, or perfect smoothness of surface, gives place to a more or less abundant crop of strong hairs. This is frequently seen in association with pigmented moles, from which a bunch of hairs, commonly coloured, projects. Or the lanugo on the cheeks, chin, upper lip, neck, or bosom, or on the arms, may become gradually transformed into strong coarse hairs. There are no subjective symptoms, but the growth occasions much annoyance, even mental distress, quite out of proportion to the impression conveyed to others.

Treatment.—There is only one method of permanently removing such hairs, and it has limitations. By electrolysis, judiciously used and repeated, we can free our patients from these hairs, provided that they are not too closely set. Electrolysis causes destruction of tissue, since its action is a chemical one; and should the hairs to be removed be thickly implanted, scarring, even keloid, may follow unwise efforts to destroy them by its means. It is too powerful an agent to employ against downy hairs, or on those of the upper lip, where, the hairs being set at an angle, the destructive influence is exerted on too thin a portion of the skin. The straighter the hair, the more favourable for successful removal. Any good constant current battery may be employed, one of four to six Leclanchè cells suits well. The strength of current should not exceed 5 milliampères, often less suffices. The patient, reclining in a comfortable position on a couch (as thus the head is most easily kept steady), in a good light, places one or more fingers in a basin filled with salt and water, in which the sponge, connected with the positive electrode, is immersed. In a needle-holder, provided with a disconnecting arrangement under the control of the operator, a No. 16 diamond needle is inserted. If the eyesight is not very acute, it is well to have a convex lens of 2½ in. focus, on a movable joint, and, sliding on the handle, to facilitate the introduction of the needle into the hair follicle. The operator holding the needle-bearer in his right hand, and a pair of epilating forceps in his left, insinuates the needle by the side of the hair into the follicle, then, closing the circuit by pressing a knob, he watches the effect. Soon a slight frothing becomes visible at the mouth of the follicle, and a blanching of the skin for a sixth of an inch round. The needle should as a rule be retained *in situ* for twenty seconds or half a minute; the circuit is then broken, and the needle withdrawn. If successful, the hair should be quite loose, and should come away painlessly with the slightest traction with the forceps. If it does not, the needle will require to be reintroduced.

The secret of success in the removal of hairs by electrolysis consists in—(1) duly estimating the depth to which the needle should be inserted, so as neither to introduce it too slightly, and thus insufficiently, or too deeply, and so cause undue reaction; (2) in determining how long the needle should be retained in position, so as to loosen the hair completely, and no more; (3) except in the case of hairy moles—where destruction of the neoplasm is intended—never to remove many hairs in close juxtaposition at the same sitting. Individual variations occur, and only experience can enable these points to be accurately gauged. The amount of pain varies in different regions and persons, it is greater on the neck than chin, with first than subsequent hairs. Not more than fifty hairs ought to be dealt with at any one sitting, nor should it exceed an hour. Thus neither operator nor patient are unduly tired, and the work is better done. A little yellowish serum exudes from the puncture, the skin feels hardened, and for a time is redder than normal. Bathing with pretty hot water, and painting with calamine lotion, serve to allay the irritation. Eventually, a faint white speck, only perceptible on close inspection, remains.

But in all cases a certain number of hairs fail to be permanently removed by the first operation, while, if the cause leading to the transformation be still at work, other lanugo hairs become strong ones. This leads to disappointment, and had best be fully explained at the outset. The method proves most efficacious in the case of pigmented hairy moles, for not only are the hairs themselves destroyed, but the mole itself flattens and becomes nearly if not wholly decolorised.

TRICHORRHEXIS NODOSA.

A greenstick fracture of the hair (Crocker). Sir Erasmus Wilson seems to have been the first to describe this, but Beigel, Wilks, and Kaposi have since directed attention to it.

Etiology.—Wilson explained it as due to defective nutrition, so that the hair is brittle, yet does not break sharply across, but after the fashion of a wet stick, first at the cortex, the attachment at the medulla persisting. Raymond ascribed it to a diplococcus which erodes the surface of the hair, and, by weakening its structure, leads to fracture. His observations were made on hairs from the labia majora of women, where trichorexis nodosa is common. Menahem Hodara has observed that an allied disease is frequently encountered in the scalp hairs of women in Constantinople, and inside the nodules he found a peculiar bacillus, with a pure culture of which he was able to infect the hairs of a previously healthy female.

Pathology and symptoms.—On some of the hairs of the whiskers, beard, or moustache, less often of other parts, there are seen a number of whitish spots, or beaded swellings, irregularly scattered over the shaft. When traction is exerted, the hair breaks at the swelling. If such a hair is examined microscopically, the cuticle is seen to be deficient at the node, the constituent fibres are dissociated, giving the impression of two brushes stuck into one another.

Treatment.—Shaving, continued for a considerable time, has been recommended, but even when this has been conscientiously carried out the disease has recurred after the hair grew. In view of the possible parasitic origin, this should be combined with the application of parasiticide remedies, such as sulphur, carbolic acid, or formalin.

MONILETHRIX.

Rhythmical thinnings of the hair shaft, imparting a beaded appearance to the affected hairs. Walter Smith of Dublin described the first case; since then many examples have been recorded, a résumé of most of which was published by Wallace Beatty and Scott.

Etiology and pathology.—Beatty and Scott have made sections of the scalp, and state that the formation of spindles begins at the papilla, and in the sections all the papillæ were forming nodes. In my case, hairs which were extracted with the root and root sheath *in situ*, first showed evidence of contraction just beyond the point of exit from the follicles. Unna is of opinion that the contraction is due to an increase of the normal elastic or muscular contraction of the hair follicle in its central region. The plastic hair mass forced through this narrow portion produces the spindle. A hereditary influence is at work in some, for M'Call Anderson has recorded a chart in which fourteen out of twenty-seven individuals were affected in six generations, and Sabouraud, seventeen cases in five. It also shows family prevalence. A similar condition may be found on the hairs generally over the body.

Symptoms.—The hair of the scalp presents a regular alternation of fusiform swellings and narrow portions. The nodes are pigmented and possess more or less medulla, the contracted parts are devoid of colour. The length from the centre of one internode to another is about 1 mm. In a case of mine, in which three children of one family were affected, by staining the hair with nitrate of silver, and shaving after an interval of four days, it was found that each node took two days to grow, which corresponded to the estimate of Smith. The node tallies in diameter with the normal, no trace of imbrication is discoverable on its surface, but the overlapping of the cuticular scales is distinct on the internode. Unna has noticed that there is spiral twisting of the whole cortex round a central axis. The hair does not attain any length, but snaps off at the thinned part. The hair, on passing the hand over it, feels harsh and wiry, and there may be coincident keratosis. All the scalp hairs are not so altered in every case, but the occipital region suffers most. The disease is a congenital one, the only exception being a case of Unna's, where it appeared to a limited extent on the vertex of a young woman after a severe fright.

No **treatment** has been suggested.

CHANGES IN COLOUR.

Various accidental discolorations of the hair arise. It may be stained blue by indigo or cobalt, green by copper, reddish brown by aniline or by the dust of greenheart timber, and purplish when chrysarobin is used, especially if washed with soap.

But in most people, as age advances, the hair becomes white. Pigment exists in the hair in two forms—one a melanotic, granular pigment, situated between the epithelial elements; the other a staining of the cells themselves. Unna thinks that the diffuse pigment disappears first. Sometimes hairs become white at once throughout their entire length, at others the root becomes white, while the older part retains its colour. While the pigment vanishes, the hair itself has a yellowish hue, that of horny substance, a condition sometimes recognisable both on the beard and scalp; but, more commonly, a silvery whiteness eventually is assumed, ascribable to the particles of air scattered through the cortex.

Greying of the hair may occur in quite young people; this is particularly noticeable in natives of the American continent, but is met with also in Britain. Some young persons have or acquire grey locks or patches, and this is hereditary in families. But besides this, individual hairs may become partially grey, so-called *ringed hairs*. On the other hand, hair which is light in childhood or youth may grow darker in adolescence, or from residence in a hot climate such as India. In all cases where the hair has suddenly altered from dark to white, the porosity is, according to Unna, altered. The normal air spaces are all enlarged, and the refractility is augmented. If there is periodicity, the hair is ringed.

The causes of senile canities are not well understood. Repeated attacks of neuralgia have been followed by more or less permanent local greyness. Great anxiety or mental disturbance has led to immediate whitening of the hair. Loss of sleep and strain influence the nutrition of the hair as of the epidermic structures in general. It becomes dry, harsh, staring, and paler in tint. An attack of typhoid fever may be the starting-point of steadily increasing canities. If the individual is young, the colour is occasionally restored. In most cases grey hair harmonises well with the complexion, and dyeing is seldom a successful expedient. There are numerous harmless methods of staining the hair practised by hairdressers.

ALOPECIA

More or less complete baldness, due to various causes interfering with the growth or renewal of the hair.

Etiology.—Though since Hebra's time the association existing between seborrhœa and baldness has been generally admitted, no thoroughly sufficient explanation was forthcoming. Unna's morococci and flask bacilli, situated in the surface layers of the epidermis, while likely enough the exciting agents of the anomalies of cornification, in the form of scaly masses more or less evident in different instances, hardly accounted, even on the chemiotactic theory, for the alopecia. The specific bacillus of seborrhœa, discovered by Sabouraud appears to occupy a position of greater importance. This, when it invades a follicle, occasions round it, but particularly at its base, an afflux of wandering cells. As a result of this, or of the action of toxines, the papilla wastes, producing a progressively weaker hair, and finally none at all. The sebum containing multitudes of these bacilli, with the loosened epidermic scales, constitutes the pityriasis or greasy crust. Hair after hair in succession is attacked, and, from a scalp once infected, eradication from the follicles seems an almost hopeless task, as the bacterial colony breaks up into isolated groups. While the hair atrophies, the sebaceous glands increase in size, and their secretion still shows the micro-organism. The explanation of the peculiar localisation of baldness may be that the regions first implicated offer the most favourable soil for the proliferation of the parasite. Conditions of malnutrition, particularly anæmic states, lessen the resisting power of the tissues against microbic incursion.

Alopecia is much more apt to affect men than women, the difference in the mode of dressing the hair and in the head coverings worn by the sexes may partly account for this disparity, but it has not so far been wholly explained. It does not depend on a diminution of hair-forming material in the blood as age advances, since at all epochs of life we observe the change of downy into strong hairs going on afresh.

Symptoms.—Rare instances have been recorded where infants have been born without hair, or it has not developed in later life. More frequently the failure has been partial. In the progressive form there is all over the scalp more or less scaliness, which not only occupies the spaces between the hairs themselves, but is seen as an accumulation at the root of each, and, when displaced by brushing, is rapidly reproduced in even greater amount. The hair loses its natural gloss and pliancy, falls off more copiously than it should, and is replaced by thinner, shorter, and weaker hairs. The process of thinning may commence at the crown and spread outwards and forwards, may begin at the temples and extend backwards, or in both directions at once. A tuft in front, and the growth at the lower part of the sides and on the occiput, persists longest, though it too may become more scanty. This shedding of the hair may begin in adolescence, or at any period throughout life. As a rule, the earlier it manifests itself the more rapid the advance. The denuded areas become smooth and somewhat greasy, assume a polished appearance, the skin being generally more tightly bound down than before. Itchiness to a slight degree accompanies the onset. What is known as senile alopecia is apparently the same process, though in extreme old age the hair lessens in amount everywhere as the cutaneous structures shrink.

Temporary falling out of the hair of the scalp occurs in convalescence from various conditions accompanied with continuous fever; thus it is common after typhoid and erysipelas. A patchy thinning of the hair is one of the phenomena in early syphilis. Permanent bald areas may result from lupus erythematosus, from ulceration, or from favus, etc.

Prognosis and treatment.—The prognosis of progressive baldness is not promising, but much may be done by well-directed treatment. The seborrhœa requires the management recommended under the heading. But there are certain remedies which possess a degree of stimulating effect on the hair reproduction. Among such are lotions containing chloral hydrate, resorcin, or naphthol. ℞ Ol. ricini, 60 min.; tinct. quillaiæ, $\frac{1}{2}$ oz.; resorcini, 1 drm.; sp. rosmarini, $\frac{1}{2}$ oz.; aq. destil. ad 6 oz. Or, Chloral hydratis, 2 to 3 drm.; sp. vini rect., $\frac{1}{2}$ oz.; glycerini, $1\frac{1}{2}$ drm.; aq. destil. ad 6 oz. These should be dispensed in bottles fitted with a dropping arrangement, so as to be readily applied to the scalp without wetting the hair overmuch. If β-naphthol is used, it may be dissolved in the proportion of 5 grs. to the oz. of almond oil. In those who show signs of the commencement of the complaint, careful attention is to be paid to maintaining the hygiene of the scalp; the hair after a bath should be carefully dried; hard, ill-ventilated hats, which compress the temporal arteries, starve the scalp, and encourage perspiration, are to to be eschewed, and all oils or pomades employed should be fresh and pure.

The alopecia succeeding to acute febrile diseases usually passes off if the hair is cut short, the scalp kept clean by shampooing with quillaia and water, and one of the stimulant applications above mentioned applied.

ALOPECIA AREATA.

An atrophic disease of the hair system, characterised by the appearance, usually suddenly, of circumscribed, pale, smooth, bald patches, which may by their extension produce complete baldness.

There is some doubt if the term *area Celsi*, attached to this complaint, is warranted; it is more probable that Celsus alludes to baldness in general. Sauvages seems to have been the first to mention it distinctly, but it was described and figured by Willan.

Etiology.—Various circumstances indicate that it is in some cases at least contagious. Sabouraud has brought elaborate researches to prove that the areate only differs from the chronic and progressive seborrhœal form in time, place, and degree. "A patch of alopecia areata is an acute local expression of seborrhœa." The same bacillus is found in the earliest stages, or at the advancing margin, occupying the upper part of the follicle. It disappears when the baldness is complete, and this constitutes the weak point in the argument, for why should it persist in seborrhœal baldness, and vanish in the denuded spots in the localised variety? The medium of communication has been suspected to be brushes, caps, cushions in carriages, indirect rather than direct. Any connection with ringworm, as has been argued by Hutchinson, is unproved and improbable. Both diseases may coexist, or succeed one another, yet even this is infrequent. That there is a neurotic form is shown by the well-authenticated cases which have immediately followed a fright or an epileptic fit. There are so far no criteria whereby we can distinguish the presumably contagious from the neurotic. Severe neuralgia of the region about to become bald has been noticed in a proportion of cases.

Pathology. — Whatever the cause, it is in the hair-producing structures and not in the hair, and we may compare with it the loss which follows erysipelas. The disease is one essentially affecting the papilla, leading to a suspension of formative activity. The depression in alopecia areata is not entirely due to the diminution of the fatty cushion, but to the absence of hair. According to Giovannini, it is in the main a deep-lying folliculitis; but any inflammatory phenomena proper exist solely in the first stage. There is a certain amount of œdema of the cutis.

Symptoms.—The disease commences by the appearance of one or more smooth, pale, though sometimes pinkish, patches, entirely or almost hairless, on the scalp or in the beard. These are, as a rule, roundish in shape, isolated, and form first at the nape of the neck, behind the ear, near the crown, or on the chin. The skin is soft and studded with punctate pits, the gaping mouths of the follicles. Many of the marginal hairs come out with slight traction, and their roots are tapering, withered, and twisted or bent. Much has been made of the presence here and there of short hairs, somewhat like a point of exclamation. There is no alteration in sensibility, but the skin bears stimulant remedies better than where unaffected. The denuded patches may remain little changed for a time, or progress more or less rapidly, even till not a hair exists on the surface of the body. Usually, in course of longer or shorter time, a colourless down sprouts up, perhaps falls off once or twice, the perfect reproduction not being completed for a considerable period. Even then relapses are frequent, or a recurrence of the ailment takes place after years. In the universal cases the hair may never be restored, though this has occurred ten years subsequent to the primary loss. The disease is common and apparently increasingly so.

Prognosis and treatment.—The prognosis is an uncertain one. The younger the patient, the more vigorous the constitution, the more limited the patches, and the slower the rate of advance, and in particular the more perfectly the mobility of the scalp is preserved, the better the chance of early restitution. Cases which spread rapidly, and those in which no attempt at renewal is manifest after many months, are unfavourable though not hopeless. In treatment, internal remedies exert merely an auxiliary effect, and such should be prescribed as the condition of the patient indicates, according to general medical principles. Local stimulation, however, certainly aids recovery. It has been found that by epilating the hairs for a distance of a quarter of an inch round a patch, the advance of baldness in that area is often arrested. A good stimulant is that originally suggested by Wilson—Liq. ammoniæ fort. chloroformi, ol. amygdalæ, āā ½ oz.; ol. limonum, ½ drm.; sp. rosmarini ad 4 oz. A little should be rubbed into the bald parts at first gently, as the skin bears it better, with more friction, once or twice a day. The ung. acidi carbolici employed twice a day is useful; or, if greater stimulation is desired, and the patient not under ten, the parts may be painted at intervals of a week or fortnight with pure carbolic acid. This is painful, but sometimes very efficient. The surface should be dressed with cold cream to which some boric acid has been added. Massage of the scalp, employed twice a day for five minutes, serves to prevent atrophy. To obviate recurrences, Sabouraud recommends friction with salol, 1·50, in sp. lavandulæ, 100.

FOLLICULITIS DECALVANS.

This is a subacute pustular affection of the hair follicles of the scalp spreading centrifugally, and leaving a cicatricial bald spot.

It has been described and studied specially by Quinquaud. The disease is a rare one. When seen there are usually several bald spots scattered over the scalp, smooth, white, and atrophic. On examining the marginal hairs, there is found a minute and rather indolent abscess, from which an easily extracted hair emerges. Sometimes there is little more than a small yellowish scale at the side of the hair. Except a degree of itchiness and the baldness, the complaint occasions no annoyance. In no instance which has come under my notice has it caused extensive loss of hair.

Quinquaud has found a micrococcus, to the agency of which he attributes the disease. The treatment consists in washing the affected parts with superfatted potash soap, or Hebra's sp. saponatus kalinus and warm water, and then rubbing in an ointment of sulphur and ammoniated mercury, as is recommended for the cure of ringworm.

LUPOID SYCOSIS.

A destructive usually serpiginous folliculitis affecting principally the side whiskers of adults. This was originally described by Milton, and recently by Brocq. Unna names the same condition ulerythema sycosiforme.

Etiology and pathology.—According to Unna, the atrophy of the cutis is not due to suppuration. There is a cellular hyperplasia of the cutis, to which is added an atrophy of the epithelial structures. There is marked dilatation of the vessels of the papillæ and the perifollicular network, and of the lymph spaces and lymphatics. "These exudative, acute, inflammatory changes, limited to the neighbourhood of the follicle, correspond to the periodic scaling, vesicle, and crust formation of this affection."

Symptoms.—The disease commonly starts in the upper part of one whisker, sometimes both. A narrow erythematous border encloses a crusted infiltrated area, with more or less thinning of the hair. The lesions are primarily vesicular, though ordinarily only thickish crusts are seen, or at a later stage the surface is erythematous and scaly. The disease creeps slowly but steadily downwards, causing destruction of the hairs, and finally the production of a bald, depressed scar-like tissue, over which extends a finely folded horny layer. After a time the process comes to an end, seldom quite reaching the chin. The same affection attacks the eyebrows.

Diagnosis.—The disease is simulated occasionally by tertiary syphilitic lesions of the same region. The differentiation is not always easy, but in the syphilitic imitation there are generally one or two horseshoe excavated ulcers, the crusting is thicker, softer, perhaps ill-smelling, and the progress more rapid. In lupus there are found the characteristic nodules, and the ailment is not confined to the hairy part. The final result resembles the scar in lupus, though the disease is not tuberculous.

Treatment.—The treatment best suited is that applicable to lupus. After removal of the crusts by poulticing, scraping gets rid of much diseased tissue. The hairs are then cut short, and the part dressed with perfectly fresh zinc ointment, containing 2 per cent. of ichthyol. The surface must be kept clean by washing with a superfatted soap, and a fairly strong sulphur and salicylic acid ointment made with vaseline rubbed in. Sometimes a pyrogallic salve suits better. From 5 to 10 per cent. of pyrogallic acid, and 2 per cent. of salicylic acid and ichthyol in vaseline is rubbed in once daily.

SYCOSIS KELOID.

A folliculitis affecting principally the nape of the neck, leading to the development of a fibroma of a keloid type, was described as dermatitis capillitii papillaris by Kaposi, then by Bazin as acne keloid. One of the best accounts of it was furnished by Morrant Baker.

It attacks the back of the neck and also the scalp, rarely the bearded face. There are circumpilar papulo-pustules, which commence insidiously, and are small and superficial. In place of healing and disappearing, a nodule forms in the skin, persists and is succeeded by others involving the neighbouring follicles. Thus the process spreads, the pus and nodule formation continually advancing farther and farther. The skin over the part assumes a pinkish or ivory white aspect, and a gristly, uneven growth is produced, which may be firmly bound down. The hairs which remain are grouped together into bunches like a brush, and are abnormally deeply set. Pus wells out on pressure from the openings, and thick, malodorous crusts are apt to form.

Keloid, as is well known, often originates in acne scars, and extends from the point of commencement, here a similar process immediately follows on the folliculitis. Ehrmann, as the result of cultivations, concludes that the ordinary organisms of suppuration are present. The keloid is probably due to some idiosyncrasy. Unna, however, states that as impetiginous pustules or true comedones are not present, the affection is a distinct one. There is a perifollicular inflammation implicating the entire follicle, and accompanied by the formation of fibrous tissue round it. All the shorter and weaker ones atrophy, only the deepest resist. These are pressed together, and their necks unite in a common infundibulum. From these a brush of hairs usually projects.

The recognition of the disease should be easy, its characters are so peculiar. It has no tendency to spontaneous cure, though after a time the hardness diminishes. Ehrmann has succeeded in putting an end to the suppuration and in obtaining a smooth and soft scar by the persevering employment of electrolysis. The same method was had recourse to as in the removal of hairs, but the needle was introduced more deeply, and retained longer in position.

SYCOSIS.

A purulent inflammation of chronic type, affecting those regions where the hair follicles produce closely set, thick hairs. The disease has long been known, but we owe to Bockhart the recognition of its true nature. Though most common on the bearded face, a parallel condition is found on the eyebrows and eyelids, in the axilla and on the pubes.

Etiology and pathology.—A chronic nasal catarrh frequently precedes the onset of that form attacking the upper lip, the morbid secretions from the nose, the vibrissæ being sometimes in like manner affected, infecting the hair follicles of the moustache. On the chin or cheeks it may arise from contagion, as by being shaved, or by inoculation if the beard is worn. At times eczema seems the starting point, or a ringworm of the beard may eventuate in sycosis. The efficient agent, as shown by Bockhart, is the staphylococcus. Unna describes four stages. In the first, which corresponds with the implantation of the cocci, the pustulation is limited to the follicle neck, followed in the second by a nodular perifolliculitis. The third is characterised by somewhat deeper suppuration, not only involving the follicle, but the cutis round it. The fourth is not constant; it consists in complete suppuration of the entire follicle, and the permanent destruction of the hair. All the follicles in the region do not contain cocci, but are involved in the inflammatory œdema of the intervening tissue. It is this œdema of the cutis, constricted at the follicular necks, which produces the "raspberry-like" translucent red granulations sometimes seen.

Symptoms.—It commences by the appearance, either on the chin or in the moustache in the centre of the upper lip, of small pustules at the point of emergence of a hair. These may be scattered irregularly or aggregated into patches. They are seated on a reddened and painful base, since the inflammation extends beyond the pustule into the surrounding tissue. Sometimes there is no great amount of infiltration of the integument, but often this is much swollen, œdematous, uneven, and nodulated, or even fungating, covered, if the hair is any length, with thick greenish or brownish crusts. Removal of the hair by epilation is usually very painful, and it brings with it the root sheath, which is spongy and translucent. On pressure, pus may exude from the follicular neck, or drops of a gummy fluid appear. The form which attacks the upper lip may remain long limited to that locality, and may exist alone, but that affecting the chin or cheeks tends to spread to adjoining parts; or fresh areas of disease may arise independently. The complaint is an obstinate one, lasts for years, at times better at times worse, but never wholly disappearing. It may be complicated by, sometimes develops out of, an eczema or seborrhœic dermatitis. Though encountered in those apparently robust, many present the strumous type.

Diagnosis and prognosis.—The features presented by sycosis are so distinct that little difficulty should be experienced. In ringworm of those regions, the diseased hairs are, as a rule, loose in the follicle, and itchiness rather than pain accompanies it. The most certain test is to search for the trichophyton in some of the loose hairs, choosing such as have but little suppuration round them. Favus rarely attacks these parts, the hairs are then firm, and the achorion can be seen ramifying within them. No promise of rapid cure can be held out in sycosis, under the best devised measures progress is slow, and the complaint specially liable to relapse or recur.

Treatment.—The disease must be conquered rather by mild and persevering treatment than by heroic remedies. The hair should be cut quite short, then poultices of cold wheaten flour starch containing some boric acid should be continuously applied till all the crusts have been removed and the inflammation reduced. The hair being kept short by shaving, the parts are to be painted daily with a solution of nitrate of silver in sweet spirits of nitre, in the proportion of 10 to 16 grs. in an ounce, the poulticing at the same time being persisted in. All crusts or accumulations are to be removed by washing with superfatted soap and warm water. If it is necessary that the patient must go out, this treatment may be carried on at night, while in the day time an ointment of salicylic or boric acid 10 grs. in an ounce of cold cream is smeared thinly on. Another plan is to apply the ung. hydrargyri ammoniati, full strength or diluted with vaseline on strips of cotton, secured in place by a mask of layers of muslin impregnated with a hard glycerin jelly. The formula for this is—zinc oxide, ten parts; gelatin and water, each thirty parts; glycerine forty. Sulphur has a reputation in sycosis; it may be used as an ointment containing half a drachm of sulphur, 15 minims of creasote, and an ounce of lanoline or vaseline. This is well rubbed in once a day. The beard must not be allowed to grow till at least a year after complete cure, else the disease will almost certainly relapse. Nasal catarrh must, if present, be treated by syringing with warm solutions of salt and water, careful drying, and the application of salicylic vaseline. If the margins of the eyelids are affected, all crusts are to be gently taken off, and an ointment of 1 gr. of yellow oxide of mercury, a drachm and a half of lanoline, and 15 minims of almond oil and distilled water applied night and morning.

KERATOSIS FOLLICULARIS CONTAGIOSA.

Syn., Lichen Pilaris; Lichen Spinulosus.

A disease of the hair system, though partially affecting the sweat glands also, in which groups of dry, reddish, pin-head sized papules form, with a projecting horny spike in the centre. Cazenave apparently was the first to mention it as acné sebacée cornée. Hardy, Wilson, Lesser, and Morrow have referred to it, but Brooke alone observed the contagiousness.

The ailment is rare, and chiefly seen in children. It may develop rapidly in crops, or extend more slowly by progressive advance. The skin becomes dry, and minute black specks appear, generally three in number, and arranged in line. Some remain stationary, and lose their dark colour; others rise from the surface as a red acuminate papule, from the apex of which a horny spine projects. Some have a more fleshy appearance, but all are discrete, and may be set with mathematical regularity. To the hand passed over them the sensation is that of a nutmeg grater, and if the horny peg be turned out, a depression is left in the papule. When fully developed, the whole surface has a dirty yellowish brown hue. The regions principally affected are the back of the neck, the outer aspect of the arms and thighs, the abdomen and popliteal spaces. It may also, though not often, attack the face. Untreated it may persist indefinitely.

There is hyperkeratosis not limited to the follicle, accompanied with slight congestion, then plugging with the formation of horny cysts or plugs. No organisms have been discovered, and the cause is unknown. Brooke has clearly traced its communicability to several members of a family. It resembles keratosis pilaris, the minute acuminate epidermic papules so apt to form on the outer aspect of the upper arm or thigh, but in it there is merely a corneous accumulation at a hair follicle, but no spine, while the condition is diffuse.

Treatment consists in washing with moderate friction, either with a resorcin and salicylic soap, or with Hebra's spiritus saponatus kalinus, and warm water.

THE NAILS.

THE nail may be aptly compared to a flattened hair, growing from the matrix at and beneath its proximal end, attached to but sliding over its bed, covered in at its root and sides by infoldings of the skin, and only free for a short space at its distal termination. Though the nail-plate may undergo morbid changes, these are for the most part secondary to affections of the matrix or bed.

The nail may not be free at its distal end, as in a case reported by Ramsay Smith, possibly due to a persistence of the eponychium. The epidermic fold behind the lunula may extend too far forward, requiring repression. The longitudinal striæ may be unusually marked, leading to a coarseness of aspect. It may become spoon-shaped, the plate being thinned and concave from side to side, with the edges everted. Wasting diseases may produce this deformity, but this does not explain it in all cases. Transverse furrows form and persist till by growth they disappear at the point of the finger. Such are occasioned by some severe illness, where there is deficient epidermic production. White spots are developed owing to the existence of air between the lamellæ. Or the entire nail may be white, as in the cases related by Giovannini and Unna.

ONYCHITIS.

Affections of the nails due to particular skin diseases will be found under the appropriate heading.

Owing to a chronic symmetrical inflammation of the matrix and nail-bed, the nails become opaque and thickened. Their edges and surface break up, and assume a rugged, dirty, and fissured appearance. The nail substance is soft and spongy, the nail-bed tender and bleeds readily. The cause is obscure, since this arises independently of any cutaneous eruption.

The chronic symmetrical onychitis is little amenable to treatment. Constitutional tonics are called for, while the nails themselves should be kept enveloped in strips of lint soaked in peroxide of hydrogen, and covered with guttapercha tissue.

ONYCHIA MALIGNA.

This commonly only involves one nail, and is found in association with evidences of struma elsewhere, or can be traced to direct tuberculous infection. The end of the digit is swollen, clubbed, and duskily congested. The nail-bed is converted into an angry-looking sore, over part of which is found the discoloured, loose, and recurved nail. There is suppuration with sanious discharge, accompanied by heat and throbbing. Though healing and the formation of an imperfect nail may ensue, a severe form of whitlow occasionally results. In onychia maligna the loose nail should be removed, the surface cleansed and soothed by the application of poultices of borated starch, applied cold. Afterwards the salicylic vaseline may be tried, or a 10 per cent. pyrogallic acid ointment in vaseline, applied for three or four days, followed by a freshly made zinc ointment containing 2 per cent. of ichthyol.

ONYCHOGRYPHOSIS.

In this disease the deformed nail bears some resemblance in miniature to a ram's horn. The growth replacing the nail extends forward and upwards, is ridged both in its length and crosswise, is shining on its surface, and of a yellowish or brownish hue. Beneath is found a quantity of soft, often fœtid substance. This is usually seen on the great toe. It consists, according to Virchow and Unna, of a keratoma of the nail-bed, combined with keratoid hypertrophy of the nail-plate. The cause appears to be external pressure, exerted by the ill-fitting, short, or tight shoe on the ball of the toe. This starts the process, which continues long after the original source has ceased to be influential. "The best examples of gryphotic nails are met with in old bedridden people." Onychogryphosis needs softening and removal of the keratoid structure, and the application of an astringent to the nail-bed, such as perchloride of iron, or a little nitrate of lead in powder.

ATROPHY.

Atrophy of the nail causes the plate to become abnormally thin, it breaks off ere the point of the finger is reached, and the epidermis creeps over the denuded nail-bed. Sometimes a suppurative process advances from the tip backwards, thinning and separating the nail-plate. This seems due to infection by pyogenic organisms, which have insinuated themselves under the nail.

INFLAMMATORY AFFECTIONS.

ERYTHEMA.

UNDER this general term it is necessary to group a number of skin affections, having as their one common feature, that there is hyperæmic congestion, disappearing more or less completely on pressure. The localised vascular disturbance may be quite superficial, or inflammatory phenomena may be combined with this, giving rise to other and deeper tissue changes. The designations "active" and "passive" have been applied to the appearances in question, but for these Unna substitutes "congestive" and "stagnatory" as more accurate. In the former there is excess of blood in the vessels, with diminished resistance and increased rapidity of the blood stream; while in the latter there is an over-filling of the skin vessels, with increased resistance and diminished speed of the stream. The darker and bluer the erythema looks, the greater the filling of the deeper-lying veins and arteries, which have a bluish tinge through the lighter red of the superficial vessels. Stagnatory congestion is apt to complicate the simpler hyperæmia. Then œdema and pigmentation may manifest themselves.

Of what may be called *simple erythema* the eruption produced by chloral in certain individuals may be taken as a type. There are deep red blotches which come out on the hands, the face, or about the larger articulations. The duration, though usually transient, is not invariably so, and while subjective sensations are as a rule absent, there may be itching and tingling. To make the parallel more complete, in a case of Köbner's there was an erythema exudativum which spread over a great part of the body. Morrow regards it as an angio-neurosis caused by a paralysis of the vasomotor centres of the head and neck. Erythema indeed is one of the most frequent of the eruptions induced by drug intoxication.

Under *erythema fugax* are included a variety of superficial rashes seen chiefly in infancy, often set up by disturbances of digestion, or during dentition, and due to the readiness with which reflex action expresses itself on the child's integument. The red is patchy, appears suddenly, and its duration is transitory.

From the apposition of two surfaces in fat infants or adults, the heat, moisture, and friction give rise to a superficial redness. A mucoid fluid becomes exuded, which in decomposing scalds the parts, rendering them hot and painful, while a penetrating odour exhales. To this the term *erythema intertrigo* has been applied. In adults this must be diagnosed from eczema, though it may be transformed into it. In infants it may be mistaken, when situated about the nates, for the eruption of congenital syphilis. Careful search should therefore be made for other symptoms of syphilis, as snuffles or mucous patches. The soles too are often dry, brownish red, and cracked in the latter.

Great cleanliness as to washing and napkins must be observed in infants, though soap is to be used sparingly, and the parts well dried. A dusting powder of boric acid 10 per cent. in talc, is advantageous in some instances, in others a weak boric or salicylic vaseline or cold cream suits better, and forms a more efficient protection. In adults the parts may be painted with a solution of nitrate of silver 10 grs. in an ounce of sweet spirits of nitre. This dries up the exudation, and the powder or ointment is more rapidly curative.

When the lower limbs are much swollen, especially in cardiac or renal disease, the skin becomes red, tense, and glistening. The epidermis may rupture or vesications form. To this the name *erythema læve* used to be applied. Rest with elevation of the leg is urgently demanded, while remedies calculated to reduce the œdema are employed.

Erythema solare or sunburn is the redness produced sometimes by the direct rays of the sun, frequently by reflection from water or from snow, especially that recently fallen. Conversely *erythema pernio* or chilblain denotes the patches of redness which occur on the hands, feet, or ears of those who have a feeble peripheral circulation. These are chiefly seen during raw, cold, frosty weather, or when the temperature suddenly alters. They are reddish or purplish blotches, appearing on the heels or toes, causing the shoes to feel too tight, or on the fingers, rendering them stiff and painful. Itching is experienced at particular times of the day, in changes of weather, as a thaw, or on entering a warm room or partaking of hot viands. If neglected they inflame, blisters and ulcers form, which heal with difficulty. In persons who toast their legs before a fire, or are exposed to the glow from furnaces in course of occupation, red patches develop, a ringed shape being assumed. When these fade, indelible brown pigmentation succeeds.

Bowles has shown that protection from sunburn is best obtained by wearing a veil of a brown colour, or by painting the face with a calamine lotion containing a little umber. Lining the hat with yellow is also advantageous. These measures neutralise the violet rays believed to be mainly causative. In those predisposed to chilblains, while tonics and cod-liver oil are advantageous, local means are most necessary. Woollen socks and gloves and broad easy boots should be worn. If unbroken, one of the most efficient applications is that suggested by Boeck of Christiania, ℞ Resorcini, 2·0; mucilag. acaciæ, aquæ, āā 5·0; talci, 1·0—*M.* This is painted on at night, forms a varnish, and usually soon cures. The application of a solution of nitrate of silver 10 to 16 grs. in an ounce of sp. ætheris nitrosi, stains the finger, but subdues the itching, and causes the chilblain to shrink. If ulcerated, wearing pieces of Unna's zinc ichthyol salve muslin on the sore induces most rapid healing.

ERYTHEMA MULTIFORME.

An acute inflammatory disease, in which lesions of very various types appear, but with the substratum of erythema. Though recognised before his time, our present conception of this complaint is traceable to the writings of Hebra. In more recent times, Begbie, Besnier, Mackenzie, Osler, and Payne have contributed to our knowledge.

Pathology and etiology.—The histopathology of erythema multiforme and nodosum consists, according to Unna, in an inflammation limited to the vessels and their immediate surroundings, with a sharply-localised œdema. In peliosis rheumatica there is extravasation of blood in addition. Views as to the etiology vary. According to some, all belong to the group of infectious disorders, the eruption being one of the symptoms. Payne suggests a relation to the erythemata produced by alkaloidal poisons of vegetable or animal origin, thus toxic. Certain facts, as the occurrence of some form of sore throat as an antecedent, the pains in the joints and limbs, and the cardiac and visceral complications, to which Osler has drawn attention, have been held to indicate a rheumatic element. The effect of cold in exciting an attack, or a recurrence, must be borne in mind.

Symptoms.—It may be ushered in by some rise of temperature, congestion of the fauces, pains in the joints, or gastro-intestinal disorder, or malaise, or some or all of these are absent. The seat of predilection for the eruption is the backs of the hands, the arms, face, feet, and legs, less often the trunk. The symmetry is considerable, though not absolute. Subjective sensations are slight. It has in most of its forms a decided tendency to recur, often at particular seasons, or may occur in small epidemics. A gradual rise from the simpler to the more complex lesions, from the superficial to those which implicate the deeper tissues, can be traced. The duration in most instances runs from two to four weeks, but some may last months or even years, little modified.

Distinctive names have been employed for varieties. The least complicated are *erythema papulatum* and *tuberculatum*. The spots are rounded, elevated, flattened, from pin-head to a threepenny piece in size. The colour is rosy, passing later into a purplish or violet shade, especially in the centre. This type is specially localised on the back of the hands and wrists. An involution of the middle of the erythematous spot may occur, with marginal extension, giving rise to *erythema annulare*. Should the circles so produced encounter others, the eruption fades at the point of contact, and gyrate lines appear. At times a series of concentric rings arise, which may exhibit alterations in hue from yellow to red or purple, imparting an iridescent aspect, *erythema iris*. The opalescence is due to the stagnation of blood in the deeper veins shining through the more superficial red, partly also to difference of age in the circles. Recurrences are frequent in this form, under the influence of cold or season. In all there is some œdema of the cutis and dilatation of the lymph spaces, but this may go on to vesiculation or even the formation of bullæ. The serum contained becoming absorbed in the centre, an appearance resembling a vaccine vesicle results. This has been called *hydroa*. Should such vesicles form on the rings of an erythema iris, the condition termed *herpes iris* is developed.

The disease known as *peliosis* or *purpura rheumatica* or *thrombotica* is but a phase of erythema multiforme, modified by some alteration of the blood or peculiarity of the tissues. The same constitutional symptoms precede the hæmorrhages as are met with in erythema multiforme of the more ordinary type, or these may be absent. There are maculæ or patches either level with the skin or slightly elevated. Such, however, are bright red at first, passing eventually into brown, do not disappear on pressure, and run a course of three to six weeks. They primarily appear on the lower limbs, but may occur on the arms, seldom on the trunk. They are not abruptly margined, and thrombosed vessels may be seen near. Relapses occasionally happen, prolonging the complaint for months, and typical lesions of erythema may be mixed up with the hæmorrhages. The effusion is always superficial, in the cutis, but beneath the level of the papillæ. While the pains and cardiac complications serve to ally this with rheumatism, it is not unlikely that there is a scorbutic element present also.

With the same local erythematous characters, we sometimes encounter cases in which a necrobiotic change takes place in the epidermis. To this the term *erythema gangrænosum* has been attached. It is the opinion of many that these should be relegated to the feigned or artificially induced eruptions. It is true that such have been almost exclusively found in females of a hysterical class. We meet with eruptions in such persons due to carbolic acid or mezereon, yet when these are eliminated there are still a residue where intense erythema in non-accessible situations appears, followed by a superficial epidermic slough.

In *erythema nodosum*, the constitutional symptoms are more severe. In the prodromal period, lasting from one to five days, there is complaint of malaise, feverishness, and weariness on exertion. Then firm, oval, somewhat elevated, extremely tender, reddish or purplish nodules seated *in* the skin, appear suddenly on the shins, arms, or even the face. The nodules are shining and tense, and look as if they would suppurate, but never do. Each lasts from a week to ten days, passing through the shades of colour of a contusion. Successive crops may come out, thus prolonging the total duration. Recurrences are rare, but not unknown.

Bazin, in 1860, described a form of erythema now designated *erythema induratum*, but his description did not attract the notice it deserved. Recently Besnier and Feulard, Elliot, Colcott Fox, Hutchinson, and Patteson, have brought it into prominence. It is more often observed in females, either near puberty, or in older persons who stand long at work, than in males. Many are lymphatic, or present the scrofulous type; they are out of health, with a feeble circulation. The lower portions of the legs, particularly the backs and outer parts, are its special situations, but it has been seen on the knuckles and elsewhere. It commences by the formation of deep-seated pea or nut-sized indurations under the skin, felt, but not visible. These, which are not particularly painful, even to handling, slowly encroach on the skin, and eventually produce dusky red or purple projections. These may be absorbed, or break down into punched-out ulcers, not unlike tertiary sores. Some doughy œdema accompanies them, and the complaint is favoured by cold weather. It may be mistaken for gummata, but its long duration, the age at which it more usually occurs, and its not being benefitted by specific treatment, assist in coming to a decision. Iodide has rather a prejudicial influence, increasing the tendency to ulceration. The tenderness on pressure, and the spontaneous disappearance of the swellings in erythema nodosum will discriminate. Prolonged rest in bed, the administration of tonics, and a nutritious dietary, with careful bandaging, and the local application of an ointment of bisulphide of mercury, 4 or 5 grs. in the ounce, lead to a somewhat protracted cure.

Diagnosis.—In diagnosis, lupus erythematosus may cause confusion; but this is seldom limited to the hands; it usually occurs also on the face and lobes of the ears, and on the patches are found rough splintery scales not met with in erythema. Lichen planus occupies the same situations, but the crimson red, glancing, often angular, itchy papules are not like erythema. The multiform character of the lesions, the acute onset, the symmetry, and the seasonal recurrence, are the features to be relied on as peculiar to erythema multiforme.

Treatment.—Rest in bed is desirable in most forms of erythema nodosum; to this should be added painting the elevations with flexible collodion. In other types the application of calamine lotion seems advantageous. Internally, salicin, or the natural salicylate of soda, or quinine as originally recommended by Trousseau, and recently by Payne, relieve the pain, lessen the pyrexia, and act beneficially on the cutaneous lesions. In peliosis rheumatica, ergot and aromatic sulphuric acid are of value. Watch should be kept for cardiac complications or nephritic ones, occasionally observed.

URTICARIA.

An eruption consisting essentially of wheals, accompanied by itching or tingling sensations.

Etiology.—The etiology is sometimes direct, and due to animal parasites, or the irritation of poisonous plants. Often indirect and reflex. Many articles of food, in predisposed persons, occasion it; various medicines, among which may be named quinine, salicylic acid, and copaiba. Worms in children are a source, while there are remote uterine or ovarian influences at work after puberty. There is reason to believe that it is kept up in some by gastric or intestinal fermentation, and by the gouty diathesis.

Pathology.—The anatomy of the wheal has been worked out, among others, by Unna. The lymph given off by the capillary system of the papillary portion of the dermis is, in the main, again taken up by the large cutaneous veins. Should these be spasmodically contracted, the lymph movement in the skin is arrested, accumulation takes place, and the meshwork is distended. We must assume, according to Jacquet, a particular condition of the cutaneous vasomotricity, and a local excitation, which provokes the neuro-paralytic reaction.

Symptoms.—The familiar wheal produced by the stinging nettle may be taken as the type. In some persons wheals can be evoked at any time by friction; in those suffering from nettle-rash, one can sometimes so provoke them as an aid to diagnosis. The wheal itself is a solid, softish or firm, flattish elevation, at first uniformly red, but afterwards presenting a more or less extensive whitish centre. Though small to begin with, each may rapidly enlarge, and, by the coalescence of several, wide areas may be involved. The shape may be round, linear, or irregular. While individually as a rule, evanescent, the duration of the disease being prolonged by fresh outbreaks, they may occasionally persist for days or still longer periods. The associated burning or tingling is an almost constant feature. Not only may any part of the integument be attacked, but the internal mucous membranes may be similarly affected. The complaint usually asserts itself suddenly, though, in what has been termed the acute form, some constitutional disturbance may precede its actua lonset. In the chronic variety, the continuous, though irregular, evolution of wheals goes on indefinitely, the slightest external irritation serving to bring out a crop.

There is a close connection between the wheal and the inflammatory papule, and what is known as *urticaria papulosa* or *lichen urticatus* is a combination of the two. The wheal, according to Colcott Fox, is primary, with a hardish spot in its centre, as is seen after an insect bite, which persists after the subsidence of the wheal. This constitutes a common form of cutaneous disease in infants, or older children, brought to dispensaries. On the trunk and limbs are seen small, flattish wheals, with numerous pin-head-sized, red, hard papules, or minute vesicles, torn to some extent by scratching. It begins about the third month, and may last till the child is two or three years old. The skin is waxy and bloodless, often thin, dry, and ill cared for, with little subcutaneous fat. The itchiness is most annoying at night, and while parasites may cause some cases, they can be excluded in many.

Though, in describing their symptoms, patients frequently employ the word "blister," it is only occasionally that a vesicle or bleb develops on a wheal. In other cases hæmorrhage takes place into it, giving rise to *purpura urticans.* What is certainly closely allied to urticaria, though the subjective sensations, due to the depth of the lesions, are often little marked or absent, is *acute, circumscribed, cutaneous,* or *angioneurotic œdema.* In this there appear œdematous tumefactions of the skin, and subcutaneous cellular tissue, from half an inch to several inches across. These are met with on the face, particularly in the neighbourhood of the eyes, or about the lips, in the vicinity of the joints, or on the trunk. Such may be soft or firm, reddish or pale and translucent, and accompanied by a feeling of tension. They, too, may form in the throat or larynx. While developing suddenly, they do not reach their full size for hours, and resolve with equal tardiness. Gastro-intestinal symptoms may prelude their formation; they are seen in association with rheumatoid arthritis, while a chill may be the immediate starting-point. Once having occurred, they are apt to reappear.

Diagnosis.—This must often be made in the absence of any lesions. The description given of red evanescent patches, or "blisters," accompanied by the sensations above mentioned, is usually sufficient to identify it. It is to be borne in mind that urticaria may complicate or form a part of more complex eruptions. Thus it is met with in pediculosis and dermatitis herpetiformis. Lichen urticatus resembles scabies and pediculosis.

Treatment.—In acute or recent cases, a timely emetic, or smart, non-irritating purge, may at once and entirely rid the system of the *fons et origo mali.* In chronic cases the dietary and habits are to be carefully investigated, and any idiosyncrasies inquired after. Sources of local irritation may be discovered. The urine should be thoroughly analysed. In cases of no long duration, neutral citrate of potass, with or without the addition of small doses of colchicum, should be given, in free dilution, between meals. Warm baths are to be avoided, but the skin may be dusted with emol keleet, which possesses considerable antipruritic power, or with talc and camphor.

In chronic cases the original exciting cause may have ceased to be operative, yet a morbid irritability of the skin persists. Listerine, five to ten drops thrice a day, in a wineglassful of water, or sulphurous acid, from half to a drachm in a like quantity, are of value as arresting gastro-intestinal fermentation. Salicin, natural salicylate of soda, and quinine, even when no malarial element is discoverable, are useful. Sulphate of atropia, $\frac{1}{120}$ gr., in pill at night, or 5 minims of ichthyol, well diluted, twice a day, are valuable in protracted instances. Locally washing with menthol soap, and the application of a lead or carbolic and spirit lotion, will be found to diminish the irritability of the integument. Lichen urticatus in children needs an improved dietary, emollient baths of warm water, containing starch or bran, washing with naphthol soap, and the use of a naphtholated vaseline, 10 to 15 grs. in the ounce. Antipyrine, in doses to suit the age, and given at bedtime, is of advantage.

URTICARIA PIGMENTOSA.

This is a disease commencing in early childhood, in which, besides wheals, there are solid, deeper, and more persistent lesions of the skin, leaving long-standing pigmentation.

The first case was recorded by Nettleship. Tilbury Fox described and figured it as xanthelasmoidea. It may appear soon after birth, seldom later than the third month. Besides wheals, there are red or pink raised patches, conical at first, but becoming flattened, and changing to yellow or brown. The surface of the elevations is somewhat wrinkled, and they feel firm. The trunk and limbs are chiefly affected, but the face does not escape. The peculiar persistence in the same situations for indefinite lengths of time is characteristic. The child seems spotted over with fairly symmetrical patches, varying, according to their age, from bright red to brown. Fresh lesions come out at varying intervals, and intense itchiness accompanies it. As the child becomes older, the eruption ceases to progress, and, after puberty, it fades in most cases. In addition to œdema of the papillary portion of the cutis, Unna has shown that there are found an abundance of mast cells, and increased pigmentation of the basal layer of the epidermis. The only disease which resembles it is xanthoma multiplex. The history of early commencement will in general serve to distinguish. Treatment has proved unsatisfactory. Crocker recommends salol in suitable dose. Antipruritics, useful in urticaria, will prove valuable here. It is essential that the condition of the bowels, as to regularity, should be carefully attended to.

PRURIGO.

A disease with an urticarial basis, dating, in most cases, from infancy, but characterised in its full development by the presence of pale-coloured papules, chiefly on the extensor aspects, accompanied by intense itching. Though a mild form was recognised by Willan, yet it is to Hebra that we owe the identification of the severe variety, cases of which occasionally, if rarely, are met with in this country, the home being particularly in Austria.

Etiology.—The etiology is unknown; it is a disease, in the main, of the poor and ill-nourished. Its urticarial origin seems well established, this having been worked out by Riehl. It is something more than a "pruriginous dermatitis in individuals, with an idiosyncratic susceptibility and a deficiency of self-control." It is subject to spontaneous remissions, but can only be cured if promptly and judiciously treated in its early stages.

The anatomy of the prurigo papule is still not wholly determined. The latest observations, those of Unna, point to a spastic œdema of the cutis, and an increase in the perithelia of the vessels. With a central pronounced cellular infiltration of the cutis, there are superficially-placed epithelial changes, consisting of proliferation at the hair follicles, and circumscribed areas of softening and necrobiosis. Leloir and Tavernier described cystic cavities in the prickle layer. These areas of softening probably give rise to the itching. The scratching eventually leads to hardening and thickening, and, by pressure, to the absorption of the fatty cushion.

Symptoms.—Commencing in early infancy with the features of lichen urticatus, the tendency of the wheals is, with the increasing age of the child, to become individually smaller, and to locate themselves more particularly on the extensor surfaces of the trunk and extremities. When three or four years is reached, only papules, felt in the skin rather than visible to the eye, and from the size of a millet to a hemp seed, arise, wheals developing solely during severe exacerbations. These papules, which, if sometimes pink, are more often of the normal hue of the skin, are found especially on the outer aspects of the legs, the front and outside of the thighs, the forearms, and back of the upper arm. The loins and gluteal regions are also affected, the trunk, and, to a slight degree, the face; but the flexures, as the ham and axilla, remain free or little involved. The papules may be few, or so numerous as to convey the sensation of a nutmeg grater. Itching is intense and wearing. The scratching occasions thickening, dryness, and powdery desquamation; pigmentation is produced, the hairs on the body are broken off; that of the scalp is lifeless and dusty. The inguinal glands are swollen into large, doughy masses. The heat of summer, in countries where the alternation is marked, leads to an amelioration; but this is not so definite, in all cases, as to attract attention. Persons who have long suffered from prurigo—for, once established, it exhibits no proclivity to spontaneous cure—have an anxious, worn look; their integument is wrinkled, and the subcutaneous fat is much diminished. There is malnutrition, from sleeplessness, the result of the intense itching.

Diagnosis and treatment.—If the characteristics notified are borne in mind, there should be little difficulty in establishing the diagnosis. As Crocker remarks, it is most likely to be mistaken for a severe chronic eczema in a xerodermatous subject, an eczema engrafted on an ichthyosis of a mild type. But there are none of the peculiar papules, and no history of urticaria. It must not be forgotten that a somewhat similar condition may develop after the period of childhood, and in which the urticarial element is absent, or slight. In such we should be careful to eliminate all local sources of irritation before coming to a decision. In the treatment of prurigo, an easily assimilable and nourishing dietary should be formulated, in which fatty materials, as cream, butter, and cod-liver oil, must be included. With this, rest in bed, the administration of warmish baths, containing carbonate of potass, or sulphuret of potass, followed by the inunction of a 2 per cent. to 5 per cent. salicylic vaseline, will be found advantageous in many cases. The itching is lessened if cannabis indica, as suggested by Crocker, in doses up to the limits of tolerance, is administered. Antipyrine is also useful in the case of children, given at night. In long-standing examples, the subcutaneous injection of pilocarpine, while the sufferer is in bed, and its action promoted by copious draughts of warm water, has proved most beneficial. β naphthol, from 2 per cent. to 10 per cent., in vaseline, acts well as an antipruritic. Glycerin of starch, made with double the pharmacopœial quantity of glycerin, with the addition of 10 grs. of boric or tartaric acid to the ounce, is cleanly and soothing. This should be rubbed on, then allowed to dry spontaneously.

ECZEMA.

A catarrhal inflammation of the skin, presenting various types of eruption, and different stages, in which the exudation of serous and cellular elements is a marked feature; and in maintaining, possibly in originating some of its forms, parasitic agents play a part.

Etiology.—Only general statements can be ventured on as to the cause of eczema. There are individuals who seem to be eczematously disposed, since very slight disturbances, either systematic or external, serve to induce attacks or recurrences. Though the subjects are apparently in good health in many cases, in others they are thin, pale, and ill nourished, with less subcutaneous fat than is normal, and in whom various lowering agencies have been at work. With many eczema is synonymous with gout, but this is too sweeping an assertion. Secondary digestion may be at fault, there may be malassimilation, and defective excretion; and this is frequently the explanation of the complaint made of debility, and disinclination for exertion. A lowering of nerve energy, a degree of what has come to be termed neurasthenia, owing to exhaustion of the reserve of nerve power, is present in some cases. Bulkley has seen the eruption appear after a period of sleeplessness. At and after middle age it is often the sign of an imminent general breakdown, a danger-signal worth attention. In infants, the child may be plump and sturdy, or thin, pale, and weakly. Teething is blamed, but it is much more an exciting than a predisposing cause.

Morbid anatomy.—The conception of eczema will probably be best elucidated by considering its pathology prior to the symptoms by which it is manifested. The morbid anatomy of eczema was very much in a chaotic state till Unna clearly formulated his views as to its histology. Though eczema develops acutely, as an inflammation associated with the formation of vesicles somewhat peculiar in kind, it is as a chronic disorder that we most commonly encounter it, and then its distinctive traits are best seen. There are then three main pathological changes, with some terminal and subsidiary ones. The first of these is an alteration in cornification, named parakeratosis, leading to the production of scales. This is essentially a parenchymatous œdema of the transitional epithelial cells, those which in the epidermis lie midway between the basal formative cells and the limiting horny ones. In the granular layer the separation of keratohyalin is for the most part arrested, but the degree varies in a punctiform manner. There is a slowing of cornification, a simplification of the process, by the omission of many of the intermediate stages. The cell wall becomes cornified or hardened, but the cell contents remain moister. The scale which results is only a hardened, improperly cornified aggregation of prickle cells. The second change is epithelial growth, acanthosis, leading to papule formation. Mitoses, those nuclear alterations which indicate epithelial proliferation, are more numerous than ordinary, and occur in abnormal situations. As a consequence of the epithelial growth, there arises a deformity of the ridge system, the projections constituting which become broader, rounder, and often elongated; while the papillæ lying between them are swollen, and here and there moderately lengthened. The third change is the spongy transformation of the epithelium, and vesicle formation. This begins as an interstitial œdema of the epidermis, a dilatation of the lymph spaces of the prickle layer. The amount of this cellular œdema increases from within outwards, so that a wide net of canals can be seen, particularly in the upper part of the prickle layer. The filaments bridging these yield at certain points, which, enlarging, constitute the vesicles. Hence the character of the exudation in eczema is primarily serous; only later does it become serofibrinous. The vesicles here described are microscopic, but visible ones may form on this basis. In one form they are nothing more than an exaggeration of the process described. But there are others quite distinct; these are relatively large, and are filled with leucocytes. Unna, in stating the differences, remarks that clinically we observe in the course of a chronic eczema, clear vesicles appear sporadically and periodically, but we also see multiple yellow opaque vesicles developing suddenly, usually traceable to an external irritant. With these alterations there are changes in the connective tissue. It does not disappear, but is infiltrated with small multiform cells, poor in protoplasm. In the interspaces between the vessels there are small spindle cells.

The imperfect cornification known as parakeratosis produces the crusts so frequently seen in eczema; these soak up the insensible exudation. If these are removed without further treatment, the condition called eczema rubrum arises. This presents the aspect of a red, moist, and swollen surface, from which serum continually oozes. There are various forms of this, as Unna has shown. In one there is so much serum that the cornification of the superficial epithelium cannot keep pace with it. There is intense spongy transformation. In another form, there is, in addition to the serous exudation, a delay in the reproduction of the epithelium. Some occult influence, possibly parasitic, is at work. The lymph spaces are packed with leucocytes. In other cases, in which the tendency to relapse is very pronounced, the prickle layer is not altogether normal, and there are remains of crusts. It has long been known clinically that, unless every trace of thickening is got rid of, an eczema is prone to recur on the slightest, even with no apparent, provocation. Unna points out that the spongy transformation has in it the seeds of persistence, so that every increased exudation, which may have no connection with the actual cause of eczema, may, after a long time, and on apparently healed eczematous areas, produce fresh crusts and vesicles.

In the eczema which attacks the palms and soles, parakeratosis constitutes the chief change. The intensity of the cornification leads to the accumulation of thick, large, and hard scales; these absorb less of the sweat secretion, which here certainly lubricates the epidermis. Hence by evaporation they dry down to the deeper layers, lose their elasticity, and deep fissures form, commencing from without inwards. Epithelial growth is relatively small, and vesicle formation limited to the upper portion of the prickle layer.

There is a form of eczema which presents many of the appearances of the true prurigo of Hebra. The complaint commences as an ordinary eczema of childhood, but, instead of disappearing, extends more and more over the extensor aspects, although not forsaking its earlier situations. The skin in such cases is anæmic, flabby, and dry; the patients scratch constantly, and as a result the horny layer becomes much thickened, and a hide-bound, leathery, and immovable condition is produced. There is, according to Unna, an increase of the cutaneous vascular tone, and the diminished supply of blood and oxygen to the epithelium which ensues favours cornification. An opposite condition of diminished vascular tone gives rise to a state in which the eczema resembles psoriasis. The epithelial growth predominates over the parakeratosis, and the œdema remains parenchymatous. Hence there is profuse production of crumbly or greasy heaped up scales, assuming a nummular arrangement, with no marked tendency to itching.

We meet occasionally with a symmetrically disposed form of eczema, in which the disease takes the form of patches in general round in shape, moist and crusted. These are chiefly scattered over the extremities, as the backs of the hands, forearms, or limbs, but may occur elsewhere. These have a tendency to swell up readily as the result of irritation, to ooze pretty freely, and are accompanied by considerable itching. According to Unna, we have here an inflammatory œdema, limited to the seat of disease. This particular form is frequently associated with nervous breakdown or exhaustion, and its appearance is a hint that, if neglected, more serious mischief is pretty sure to follow.

One of the most important and interesting varieties of eczema is that more specially associated with the name of Unna, since it is to him we owe its separation from other kinds of eczematous dermatitis. The exact nature of the process in *seborrhœic eczema* may be to some extent matter of discussion; but examples of the clinical picture drawn by Unna are of daily occurrence. In seborrhœic eczema, then, we have an increase of fat, both in the affected skin and also in the scales or crusts. Since there is nearly always some degree of hyperæmia, this imparts a salmon hue, as Brooke styles it, to the eruption. The scales themselves are often yellowish, and the skin in parts, as on the face, has a distinctly yellow tint. In itself the disease is mainly dry; a transformation into the moist condition alters essentially its characters. Hence, of the cardinal symptoms of eczema, parakeratosis is present; but interstitial œdema is lessened, while epithelial growth is pronounced. The source of the fat is possibly the coil or sweat glands in a measure, but one cannot exclude the sebaceous glands from some participation. Were the disease as well marked on the palms, where the lubrication must be effected by the coil glands, as it is in situations such as the scalp and the front and back of chest, one might admit that the coil glands alone were the source; but such is not the case. Situations well provided with sebaceous glands furnish the most typical examples.

One other point in the pathology of eczema still needs notice; this is the association with micro-organisms. Unna has described cocci arranged as diplococci, or in mulberry-like masses, his morococci, as present at least in the superficial layers of the epidermis, in many examples of eczema. Other forms, as his flask-shaped bacilli, are also to be encountered. The difficulty is that these and other organisms are also found where there is no eczema. It may be, as Payne has suggested, that from nervous disturbance, or digestive derangement of nutrition, the soil becomes prepared, and then, owing to an acquired virulence, the organisms, formerly mere saprophytes, possess the power of setting up eczema. This question is one yet far from being solved; and the same is to be said of the theory of chemiotaxis, or the attractive influence of micro-organisms on the elements of tissues. There are three principal foci from which eczema spreads. Of these the scalp is one, from which it advances over the forehead, ears, and neck. Another is located on the feet, especially between the toes; from thence the disease ascends. A third is situated in the groin, about the scrotum, and between the nates. Here eczema often persists chronically; yet, as Payne has pointed out, from thence it may occasionally spread centrifugally. It is frequently confined to the hands alone, never going further. The occurrence of pustulation, or of secondary boils and abscesses, is due to the incursion of staphylococci in an active condition.

Symptoms.—Regarded clinically, eczema is a multiform disease; so that, though there are certain characteristic features never wholly absent, yet in each example one or more comes into greater prominence. Itching or burning heat in the part is seldom awanting at some stage. Burning sensations are complained of chiefly in acute attacks, while itching may persist throughout. Itching may precede any visible sign of eczema, or it may, much to the annoyance of the patient, continue after the part has outwardly assumed a healthy aspect. Redness, due to congestion, is all but universally present. In the seborrhœic variety there is, in addition, a yellowish tint. The researches of Unna have confirmed the earlier view, which classed eczema as a vesicular disease, though the vesicles themselves may not be perceptible to the eye of the physician. As they are delicate and rupture easily, a moist exuding surface is more common, covered more or less completely with crusts. The eczematous area may be studded with hardish, acuminate, red, and itchy papules; or, in the erythematous type, there is not much more than redness, a degree of thickening, and a tendency to desquamation in fine flakes. In all long-standing cases, the skin feels thick when pinched up between the fingers, and this infiltration may extend even to the subcutaneous areolar tissue, so as to produce a considerable degree of fixation. This is often specially well marked on the scalp. The integument, thus deficient in pliancy, tends to fissure, as a result of stretching or flexion, adding materially to the discomfort.

While eczema may occur as an acute, a subacute, or a chronic ailment, these are but phases of the process, which require only to be mentioned. But the *seborrhœic form* needs more detailed description. While this principally starts from the region of the scalp, it may commence and spread from a catarrhal inflammation of the margins of the eyelids, as is not uncommon after measles; or it may begin in the axilla, groin, or perineum. On the chest or between the scapulæ, especially in those who wear habitually thick flannel undervests, and have naturally greasy skins, are seen red, narrow, crescentic, and punctiform slightly-elevated lines, including a fawn-coloured space, sometimes bearing a few flaky scales. The itching which accompanies the eruption is not very troublesome. It has a tendency to disappear and recur, perhaps at special seasons. It is often mistaken for ringworm of the trunk; but only Unna's morococci, or similar micro-organisms, have been identified. Wider areas in the axillæ, or inside of the thighs, present a more uniform redness, with a little scaliness, but not much thickening. The patches are not infrequently well defined at their margins. A more extensive eruption is encountered, wearing the aspect of a psoriasis, but not so markedly implicating the elbows and knees. The scales are more greasy, softer, and seldom heaped up to the extent customary in typical psoriasis; yet the line of demarcation is a narrow one. On the scalp the scales and crusts are thick, yellow, and often firmly attached. The scalp is, in anything like a well-pronounced case, very immovable, the hair thinned and lustreless, while the eruption extends on to the forehead, or has spread over the ears. Such a condition is frequent in infants and young adults, but may occur at any age, even on the bald scalp. A transition into a moist phase may take place, and, the characters being thus masked, the relationship to seborrhœa may be overlooked. Though exacerbations and remissions are frequent, spontaneous cure is not the rule, and recurrences after relief has been obtained by medication are common. The simplest conception of the complaint is, that it is a parasitic form of eczema, developing on a surface which has been, for a longer or shorter time, affected with seborrhœa.

Diagnosis and prognosis.—In diagnosis, other diseases are more apt to be mistaken for eczema than the converse. The variation in aspect from time to time, as a case of eczema passes through various phases, should be borne in mind. All parts affected should be compared, the history inquired into; it should be ascertained if any treatment has been employed, which may modify or alter the appearances. Lichen, psoriasis, and impetigo contagiosa are those most apt to be confused with it. Ringworm of the body, or in a chronic and diffuse type on the head, and tinea versicolor or pityriasis rosea, resemble the seborrhœic form. Scabies produces a dermatitis which may, out of good nature, be thought an eczema. The palmar and plantar forms are imitated by syphilis. The prognosis of eczema is, as a rule, good. Treatment may be tedious, and relapses are frequent, but cure is attainable in the large proportion of cases, if well-directed efforts are persevered in.

Treatment.—In the management of a case of eczema, it is of the greatest importance to eliminate all sources of irritation, which comprise accumulated secretions, scratching and friction, too stimulating remedies, lowering drugs taken with a view to a supposed purifying of the blood, or unsuitable food or beverages. The more completely we can put the skin, in whole or part, at rest, the more rapidly will we cure. No possible risk can arise from the healing of an eczema, however young or old may be the sufferer, however long-standing it may be. Before any treatment is begun, all secondary products, all crusts, etc., must be got rid of. Poultices made with starch, to which some boric acid has been added, a mild but non-irritating antiseptic, form the best application for this purpose when the disease is limited in extent. The starch should be made of the consistence of a soft jelly, should be applied cold, spread thickly on cotton, and covered on the side to be placed on the part with muslin, so as to facilitate removal. Such are on no account to be covered with an impervious material, such as guttapercha tissue, or jaconette. As each poultice is changed, the surface so exposed should be wiped rather briskly with a soft cloth, so as to remove accretions. This favours the more rapid reproduction of a healthy corneous layer. In cases of moist eczema, they tend to dry up the secretion harmlessly, and may therefore be continued for several days at least. In widespread cases, if acute, bran baths are useful; if chronic, alkaline ones. Usually 2 oz. of carbonate of potass in 30 gals. of water at 95° to 99°, or boric baths of the same strength, prove detergent and soothing, and may be supplemented by vaseline inunction. Soap is in many cases contra-indicated, but if any be employed, it should be a superfatted potash one. In eczema of the head and legs, coverings of indiarubber or oiled cloth, worn for a few days, tend, by retaining the perspiration and macerating the surface, to render the crusts more easy of removal; and the same may be said of impervious bandages for the legs, but these must be continued only a short time. In all extensive eczemas, whether wide in individual area or consisting of numerous smaller patches, confinement to bed is necessary. In eczema of the legs with œdema, the lower end of the bed should be elevated by placing blocks of wood under the feet. When hairy regions are involved, shaving is often a necessary preliminary.

The surface now freed from accretions may in many cases be best treated by the application of vaseline containing from 2 to 10 per cent. of salicylic acid; with this starch poultices may be alternated. If the eczema present the seborrhœic type, 2 per cent. or more of sulphur can often be added with advantage. A small quantity only should be carefully rubbed in once or twice a day. So long as improvement continues, no further change is required. When progress seems stationary, a vaseline starch paste, a little modified from the original formula of Lassar of Berlin, frequently suits admirably. This is composed of equal parts of vaseline, lanoline, oxide of zinc, and powdered starch, with 2 per cent. of salicylic acid, or 1 per cent. each of salicylic acid and resorcin, and 2 of ichthyol. This, to obtain the best results, should be very sparingly applied, and well but gently rubbed in. In moist forms omit the resorcin.

Sometimes better effects are produced if the ointments, in place of being smeared on, are applied spread fairly thickly on cotton or linen torn into strips. The ointments most applicable are, in oozing patches, the unguentum vaselini plumbicum of Kaposi, made by melting and incorporating together equal parts of lead plaster and vaseline. If the eczema present more of the pustular type, the unguentum hydrargyri ammoniati of half the pharmacopœial strength may be employed in similar fashion. In like manner some of the elegant salve mulls introduced by Unna, such as his zinc ichthyol salve mull or muslin are applicable. This last is a specially soothing and healing preparation, but too expensive to use over large surfaces.

When all acute symptoms have ceased for some time, various stimulant applications may be tried. Of these tar in some form occupies a prominent place. This is often best used as a lotion, as a drachm or two of the liq. carbonis detergens in a pint of distilled water, with or without the addition of a drachm of the liq. plumbi subacetatis. This may be sopped on once or twice daily. A solution of coal-tar in acetone, introduced by Sack, is even more useful, and at the same time, as a rule, unirritating—℞ Picis carbonis, 10·0; benzol, 20·0; acetone, 77·0. This, when painted on, dries into a thin yellow varnish. Carbolic acid a drachm, rectified spirit an ounce and a half, glycerin a drachm and a half, and distilled water six ounces, forms another useful stimulant lotion, especially valuable where itching persists. In obstinate examples of the seborrhœic variety, provided the affected areas are few and limited in extent, a pyrogallic ointment is of service. This is made up of pyrogallic acid (perhaps best oxidised) 10 to 25 grs., salicylic acid 10 grs., ichthyol 25 minims, and vaseline an ounce.

In the purely erythematous or papular forms of eczema, where the surface is dry, the well-known calamine lotion, with a drachm of boric acid in each 6 oz., painted on, is protective and often curative. Or, in similar circumstances, a lotion recommended by Boeck, may be tried. This is compounded of talc, powdered starch, liq. plumbi subacetatis dilutus, a one per cent. solution of boric acid in water, each one hundred parts, and glycerin forty parts. This requires to be well shaken, and diluted with a sufficiency, usually twice as much, cold water, before being painted on. It has a delightfully cooling and antipruritic effect.

There is no medicine administered internally which can be regarded as a specific. Certainly arsenic is not so, though, from the way in which it is prescribed indiscriminately, the idea seems prevalent that it is. It very seldom does any good, not infrequently harm. Saline aperients, if constipation exists, are advantageous; a good form is a wine-glassful of Hunyadi Janos water in a tumbler of warm water on rising. The action of the kidneys is in many cases defective, particularly in eczema at and after middle life. The free use of water drunk in the intervals between meals, or the administration of acetate of potass, with tincture of nux vomica and succus scoparii well diluted before food, will help to obviate this. Small doses of tartrate of antimony, $\frac{1}{32}$ to $\frac{1}{16}$ gr., given three times a day, are valuable in limiting the spread of acute and extensive forms. States of debility and anæmia are to be dealt with by suitable tonics and hæmatinics.

The diet in eczema must be regulated. It should not be too stimulating, but it must include the free use of green vegetables, well and carefully cooked. Ripe fruits are not contra-indicated. Butter and easily assimilable fats should be partaken of. Alcohol in any form ought to be taken only with meals. Tea must be taken sparingly, never long infused. Sugar in moderation does no harm, and there is no reason for discarding well-made porridge at breakfast, from any supposed heating action.

Some mineral waters at home and abroad exert a beneficial effect on eczema. Those of most value are Harrogate and Strathpeffer in this country, and Luchon, Aix-la-Chapelle, and Ems abroad. Much of the benefit is undoubtedly to be ascribed to the change, the freedom from occupation and worry, and the expectation of cure or amelioration, but the waters have also a considerable influence. Sea air is apt to be harmful in acute or subacute cases, and during an attack, or even for some time subsequent to the cure, sea-bathing may not be indulged in with impunity.

DERMATITIS REPENS.

This is a spreading dermatitis, usually following injuries, and probably neuritic, commencing almost exclusively in the upper extremities. It is to Radcliffe Crocker that we are indebted for the recognition of this rare form of disease, but additional examples have been observed by Garden, Nepveu, and Stowers.

An injury to the skin, often at a nail, seems in most cases the starting point. From it extends a dermatitis, characterised by free exudation of fluid, and producing vesicles and bullæ, which cause the separation of the upper layers of the epidermis, leaving an intensely red surface, oozing at numerous points. The border is sharply defined, consists of the sodden and undermined epidermis, and advances steadily onwards, so as to involve more or less of an entire limb. In some subacute cases the surface is dry and glazed. The nails of the affected hands or feet are gradually shed. There is no very marked itching, burning, or disturbance of the general health. In Stowers' case, however, there were burning pains in and about the affected regions, resembling those which accompany peripheral neuritis. The view of its causation held by Crocker is that the lesions are due to peripheral neuritis, set up by a trivial injury, though secondary parasitic invasion complicates it. It has been mistaken for an eczema, but the features are different. Syphilis in its later stages may simulate it, but in it there are generally separate foci, commonly arranged crescentically, influenced for good by iodide, which dermatitis repens is not. Ringworm invading the thick epidermis of the palms and soles, presents the same undermined edge, but the process is dry throughout, and the trichophyton can be found in the scales if carefully sought for. Treatment is tedious and not very rapidly satisfactory. Continuous lotions of lactate of lead cured one of Crocker's cases; painting on a 10 per cent. solution of permanganate of potass three times a day, till it formed a crust, the undermined skin being cut away before applying it, another.

IMPETIGO CONTAGIOSA.

An acute, inflammatory, contagious disease, characterised by the formation of flattish vesico-pustules or blebs, the size of a split pea or larger, drying up into crusts, which look as if "stuck on." This complaint was known to Plumbe and Bateman, and described as porrigo, but it was Tilbury Fox who first accurately identified it, and gave it the name which, though perhaps not the best, yet has come to be generally accepted.

Etiology.—Though found in adults, it is mainly a disease of childhood or adolescence. Armstrong has recently attached the designation of "football impetigo," but while it is apt to be spread through a school by players of the Rugby game, it occurs in other circumstances. The active agent of causation and transmission is, there is every reason to believe, a special staphylococcus.

Symptoms.—Isolated flat vesicles make their appearance on the face or on the occiput, sometimes preceded by a slight degree of fever. A faint areola surrounds each. Some continue small, others enlarge into flat blebs, if not ruptured by scratching. The contents, at first clear, become rapidly milky, then purulent, while the inflammatory halo fades. By the coalescence of several vesicles considerable areas are involved. Either as a result of their rupture or drying up, crusts or scabs are formed, very characteristic of the complaint. The colour of these is often like honey, at other times brownish or black from admixture of blood. On the scalp, in particular, they are hard and crumbly, and reach the size of half a nut. When removed there is underneath a superficial abrasion, exuding a gummy secretion. When the scabs have become quite dry and fall off, red stains, which fade slowly, remain. It is both auto-inoculable and communicable to others, hence it may occur on the hands of adults coming in contact with those affected, and it may attack the lining membrane of the nose or the lips. Its average duration is about three weeks, but in some cases, especially on the scalp, where pediculi are frequently present, it may last months, even in spite of a certain amount of treatment.

Diagnosis and treatment.—The diagnosis should afford no difficulty, if the features described are noted. The markedly contagious element at once distinguishes it from eczema. The eruption on the back of the head, due to scratching caused by pediculi, is quite different. There is much oozing and matting of the hair, and with the destruction of the pediculi and cleanliness the complaint disappears. Treatment is easy and efficacious. The crusts having been thoroughly removed by poulticing, an ointment consisting of 5 gr. of ammoniated mercury in an ounce of simple ointment is to be rubbed into the surface where they were, four or five times a day.

ECTHYMA.

This consists in discrete, flat, large, prominent pustules, seated on an inflammatory indurated base, succeeded by a yellowish or brownish crust. The pustules are rounded or flattened, and are surrounded by a wide and usually bright red areola. They may be dark, owing to impregnation with blood. The areola fades, and the skin wrinkles, the purulent contents drying into a somewhat bulky crust, which separates slowly, leaving a red stain or some pigmentation. There is an erosion beneath the scab, but no true ulcer, which serves to distinguish ecthyma from a pustular syphilide, as no scar is left by the former. The lesions are essentially discrete, and, while they may occur anywhere, are perhaps most common on the lower limbs or outside of the thighs.

The skin in ecthyma is dull and lifeless, often sallow or anæmic, accounted for by the circumstance that the causes are twofold. One, all those influences which lower health; hence we meet with it in tramps, the inhabitants of workhouses, or in ill-tended and ill-fed infants. Scratching is the other, which implants the pyogenic organisms, which set up the pustulation in the dry, harsh, and dirty skin.

The disease is deeper seated than impetigo contagiosa, and is only auto-inoculable. Hence the pustules and crusts are best treated by soaking in warm baths containing 2 oz. of carbonate of potass in 30 gals., or by repeated poulticing. Then a weak ammoniated mercury ointment, containing some zinc oxide in addition, should be rubbed in. All animal parasites in skin or clothes must be got rid of by appropriate means. The general health needs attention by suitable mixed dietary of meat and green vegetables. The most efficient tonics are usually combinations of dilute nitro-hydrochloric acid with quassia or cinchona.

FURUNCLE AND CARBUNCLE.

A circumscribed inflammation of the true skin or subcutaneous tissue, forming a painful, hardish tumour, softening slowly in its central portion, and discharging more or less pus and sloughs.

Etiology and pathology.—The commencement is held to be in a gland, either a hair follicle, as in the *boil*, in a Meibomian gland, as in the *stye*, or in a sweat gland, in so-called *hydro-adenitis*, but the course in all is similar. The active agent is the *Staphylococcus aureus*, but there is in most cases some influence at work which has led to lowered vitality. Sometimes the system becomes too suddenly charged with effete material, which the emunctories are incapable of getting rid of. This may be occasioned by training, too full a dietary, alteration from a limited to a richer or the converse. Other causes are found in the debility of over-lactation, in convalescence from prolonged fevers, diabetes, etc. No cause whatever may be discovered. Fresh boils are produced by auto-inoculation. Local irritation determines the locality.

Symptoms.—Though differing from each other in various points, yet *boils* and *carbuncles* have so much in common that they may very well be described together. In both, often the first thing noticed is a sensation of tension or perhaps actual pain in some part of the skin, the nape of the neck being a frequent situation, although no locality is exempt. To the examining finger there is a feeling of induration, somewhat tender to touch. This increases in size, becoming conical in the boil, flat and widening in the carbuncle. The space involved acquires a vivid red hue, or may become purplish. Pain is intensified, is piercing or throbbing, with tightness and weight. Probably the sloughing process is then advancing through the dense structures of the corium, for soon afterwards the apex of the boil, or the surface of the carbuncle in places, turns yellow, owing to the presence of pus under the thinned epidermis, while the sensitiveness to contact diminishes. Eventually one or several openings form, leading down to a greenish yellow slough. This slowly separates, being extruded through the apertures, which appear too small to permit its passage. During this process pus is being constantly secreted. While the carbuncle is generally single, boils are apt to occur in crops or in succession, new ones arising for an indefinite period of time. In the *blind boil* the tumour may resolve without suppuration, or it may not come to a head. Constitutional disturbance is present in proportion to the number of the boils, or the size of the carbuncle. The lymphatic glands near usually enlarge sympathetically, and may suppurate.

Treatment.—The local treatment of boils consists in restricting as far as possible the growth of each, and in preventing the eruption of fresh ones. This is most effectually done by applying to the boil, at as early a period as may be, a piece of Unna's mercury and carbolic plaster muslin, changing it daily or from time to time. This undoubtedly lessens the size, diminishes pain, obviates friction, and prevents auto-infection. When ripe, the contents should be gently pressed out, the part fomented, and a new bit of the plaster muslin put on. In the case of carbuncles, the continuous application of the boric starch poultice, as described under eczema, forms the most soothing dressing. This may be continued throughout, the sloughs as they loosen being gently extracted by fine forceps.

Constitutionally, after a careful review of the patient's habits, dietary, and surroundings, all sources of error should be as far as possible corrected. The influence of sewer gas and an unhealthy atmosphere must be borne in mind. Free exercise in the open air, short of fatigue, or a change to the seaside or some hilly or mountainous region, is most effective in arresting the eruption of fresh boils, and is almost essential after carbuncle. Various tonics, such as quinine with an acid, or the syrup of the hypophosphites, are indicated, and yeast, taken thrice a day in doses of ½ oz., or the sulphide of calcium, ¼ gr. in coated pill, as frequently, are empirical remedies. In gouty persons, neither diabetic nor albuminuric, Brocq has found that ½ gr. of extract of colchicum in pill daily has given good results.

HERPES ZOSTER.

Groups of vesicles on an erythematous base, arranged usually at intervals along the course of one or more cutaneous nerves. Pliny seems to have recognised zoster, speaks of the eruption being limited to one side of the body, and is responsible for the statement, not yet entirely discredited, that it proves fatal should it surround the body completely.

Etiology.—Epidemics of zoster have frequently been observed, so that in its causation some as yet unexplained atmospheric influence may be at work. A few cases are known in which there was a suspicion of infection, the disease having appeared in one or more members of a family in succession. Hutchinson and Nielsen have specially drawn attention to its occurrence during the administration of arsenic. Such have generally arisen during a first course. As arsenic is a stimulant to the active cells of the epidermis, shown by the aggravation of a progressive psoriasis or acute eczema, this may account for its power as an excitant of zoster. It has also been seen as a sequence of poisoning by carbonic oxide. Blows have been blamed for an attack, and it has manifested itself in cases where the nerves have been implicated in inflammation from diseased bones or soft parts.

Pathology.—The disease is, pathologically, a neuritis, probably a descending one, and this may be of cerebral, spinal, ganglionic, or peripheral nerve origin, since the eruption may be unilateral, bilateral, or consist of but one or two groups of vesicles, which latter could only correspond to the peripheral distribution of a nerve trunk. The motor filaments may participate, and paralysis result. The vesicles are pock-like, and, according to Unna, the special element in their formation is a peculiar degeneration and swelling of the epithelial cells, with destruction of their protoplasm, a change which he terms ballooning. "If the disease runs its rapid normal course, no scars are left. Only when suppuration is prolonged, or where from the commencement there is necrosis of a whole vascular area, with consequent suppuration, do scars indicate the seat of shingles."

Symptoms.—The first symptom is generally pain, localised to the track of a cutaneous nerve, but this in children and occasionally in adults may be absent throughout, the eruption being that which attracts attention. Only in severe cases are there any well-marked constitutional or febrile phenomena. Nausea and vomiting have been noticed at the onset of frontal zoster, with gastric and hepatic disturbance, explained by the inosculations of the fifth and eighth nerves. After a period of two or three days, red pin-head-sized papules appear in groups with an areola round each; these soon change into vesicles seated on an erythematous base. While the vesicles of each group are coeval, those nearest the central nervous system come out earliest. The contents alter; from being transparent they assume a milky and then a purulent aspect, or some or all are hæmorrhagic, in which case scars are apt to be left. If on the thorax, it is not uncommon for the eruption to extend over the territory supplied by more than one nerve. It is rare for both sides of the body to be simultaneously or consecutively affected, very rare for corresponding nerves on both sides. Still instances are on record where the eruption has been very widespread indeed. The vesicles do not readily rupture, commonly dry up, flaking off in ten days or thereabout. They may break, however, and ulcers arise which leave indelible scars. The neuralgic pain lessens or ceases with the appearance of the eruption, but may reassert itself later, and prove both obstinate and wearing. In zoster affecting the fifth nerve, should the internal branch of the nasal nerve be implicated, the cornea is apt to ulcerate; if not, there is no more than severe conjunctivitis.

Diagnosis.—The localisation of the clusters of vesicles, corresponding to the distribution of one or more nerves, is almost sufficient for the diagnosis of zoster. The premonitory pains may simulate those of pleurodynia when the thoracic region is involved, or some deep abdominal mischief may be suspected. The blisters produced by frostbite on the ear have caused confusion, and the formation of the vesicles in the hair of the occiput led to their being misinterpreted. The dilated lymphatics in lymphangioma circumscriptum much resemble zoster, but on inquiry they will be found to have been present for long unchanged, or but little.

Treatment.—Protection from rupture is what we should seek in treatment. If seen at an early period, painting the vesicles over with flexible collodion does best. If the contents are milky, or, still more, if already purulent, then we must be content to dust the part freely with a 3 per cent. mixture of salicylic acid and talc, and over this to place a covering of salicylic wool. Should any abrasions or ulcers arise, these can best be healed by applying daily pieces of Unna's zinc ichthyol salve muslin. The prodromal pain may require the subcutaneous injection of morphine, or 10 gr. of antipyrine may be given at night. The secondary neuralgia is relieved by painting with chloral camphor and enveloping with cotton-wool. If very persistent, benefit is obtained by the application of the constant electric current twice a day.

HERPES FACIALIS.

This is an eruption of small vesicles on an erythematous base, occurring on the lips, buccal mucous membrane, or lower part of the face, which tends to recur. The vesicles appear suddenly, with heat and tension, on a surface which may have previously shown a reddish blush. They form one or more groups, and, after lasting for a day or two, dry up into little scabs, which, on falling off, leave a reddish stain. Itchiness may succeed their disappearance. Sometimes there is but one blister. If the eruption comes out on the lining membrane of the mouth, the walls of the vesicle are so tender that only a superficial erosion or ulcer is seen, and this may be misconstrued. It is met with in the course of many pyrexial affections, in cases of pneumonia, or in association with a common catarrhal seizure. Hutchinson has pointed out that any disorder which is preceded by shivering is liable to cause it; he regards it as pathognomonic of a rigor. It has not the prognostic significance popularly ascribed to it.

The application of calamine lotion containing a little boric acid is protective and cooling. Balfour employs a solution of 5 gr. of kamala in an ounce of spirit of camphor, which causes the vesicles to dry up. If in the mouth, pellets of chlorate of potass should be slowly sucked, three or four times a day.

HERPES PROGENITALIS.

An eruption of vesicles on an inflamed base on the genital organs in both sexes. This is not uncommon in men, but is much less frequently encountered in women. It closely resembles herpes facialis, and like it is apt to be preceded by itching or burning sensations. It may locate itself on the inner surface of the prepuce, on any part of the glans penis, within the meatus urethræ, on the vulva, or even on the cervix.

There are seldom more than one group of vesicles, and not only is the base on which they are placed erythematous, but the neighbouring parts themselves are often swollen and œdematous. The duration does not exceed a few days, but it is specially prone to recur. The liability to reappearance, however, ceases after middle life, probably in consequence of the increased toughness and dryness of the parts. Those who suffer from it have usually had a soft sore or a gonorrhœal attack previously. In women it is seldom seen, except in prostitutes. The exciting cause of this form of herpes may be found in the congestion associated with the act of sexual congress, though, once initiated, it does recur apart from any distinct irritation of a local nature. Such recurrences are repeated at fairly regular intervals, and may continue for years, though the sequence may be broken by some illness which exerts a marked influence on the constitution. In diagnosis the only difficulty is in distinguishing an irritated or abraded patch of herpes from a soft sore. The history of a vesicular eruption having appeared previously, may aid in the discrimination. Auto-inoculation would settle the question, but this takes time, and time alone will enable a decision to be arrived at.

Treatment.—The part should be carefully cleansed with warm boric acid lotion, then dried, and if the vesicles are unbroken and still transparent, they may be painted over with flexible collodion, if not directly on a tender mucous surface. If there, or if the contents are already milky or purulent, or if ruptured, it is better to dust with a boric talc powder of 10 per cent. strength. Internally the administration of arsenic in small doses, continuously, has some effect in obviating relapses.

PEMPHIGUS.

A disease characterised by the formation of bullæ in succession, on an unaltered skin. We owe the term pemphigus to Sauvages, who appears, however, to have had no very clear conception of the complaint. The morbid conditions included under the designation have been reduced by the transference of some to other categories. The disease, if rare, has distinct and well-pronounced features. Chronic in most cases, it may occur in an acute form; it affects new-born infants sporadically, or as an endemic. The bullæ may be plump, or flaccid and diffuse—pemphigus foliaceus; or, from the base of the blebs, fungous excrescences may sprout, giving rise to a dangerous and fatal variety—pemphigus vegetans.

Etiology and pathology.—Local injuries serve to determine the development of a bulla in those who are the subjects of pemphigus, and chills appear to have some influence in the production of pemphigus foliaceus; but the true cause of the disease is unascertained. There is reason to conjecture that the defect lies in the nervous system, probably in the vasomotor centres. Nor is the histology of pemphigus worked out. Unna defines the changes in pemphigus foliaceus as persistent vascular paralysis of a high degree with dilatation, especially of the subpapillary lymph vessels, and œdematous swelling of the constituents of the skin. He does not class pemphigus vegetans with the other forms, but regards it as distinct, as a locally infectious, auto-inoculable affection, leading to characteristic growths like condylomata.

Symptoms.—In the form chiefly encountered, the characteristic bullæ either rise directly on normal skin, or a hyperæmic spot appears, on which the bleb forms. Individually they are from a pea to a marble in size, sometimes reaching the diameter of an egg. The contents are clear straw-coloured at first, but may become cloudy, or are purplish or dark owing to admixture with blood. The reaction is neutral or alkaline, but if a purulent change takes place, it may be acid. The walls are tense, and there is little tendency to spontaneous rupture. The fluid is not long of being absorbed, the collapsed walls form a thin scab, and when this separates a reddish stain persists for a time. The eruption may be limited to a single bleb, or to one group, or the duration may be protracted by the appearance of fresh crops, or the complaint may relapse after an interval. The constitutional symptoms may be slight or absent, in cases where the bullæ are few and the duration short. But in extensive outbreaks, or in children or aged persons, there may not only be fever and general systemic disturbance, but the prostration may be so great as to lead to a fatal termination. It is not always limited to the skin, but may attack mucous membranes, as in the mouth or vagina. When it implicates the conjunctiva, adhesion of the surfaces may occur, leading to what has been termed "essential shrinking" by Graefe and Morris. A peculiar inherent vulnerability of the skin, in which vesicles or bullæ arise in consequence of trivial lesions, has been termed *epidermolysis bullosa.*

Acute forms have been seen chiefly in children, but Pernet and Bullock have recently drawn attention to a dangerous and fatal variety met with in butchers, or in persons engaged in handling meat or hides. The infection probably takes place through a wound, and the cause is most likely a special micro-organism. Those in children are seen in lying-in institutions or unhealthy dwellings, affect new-born infants, and occur both sporadically and in epidemics. Such cases must be distinguished from the bullous syphilide.

The exceptional variety described and figured by Neumann, and of which an example has been recorded by Crocker, called *pemphigus vegetans,* affects the mouth and pharynx primarily, but bullæ come out on the integument in general. Ulcers and fungating growths develop in their floor, much like condylomata, and secreting a viscid and offensive fluid. The disease in this form is almost invariably fatal.

Pemphigus foliaceus was first described by Cazenave; in it the bullæ are flaccid, and the eruption tends to spread till it involves the whole body, ending fatally, though sometimes after lasting a considerable time. The fresh crops succeed each other so rapidly, that the epidermis has not time to become condensed into a uniform covering, so that the fluid poured out dries up into a coating compared to flaky pie-crust. The skin thus may resemble a scalded surface, or be mistaken for an eczema. An extremely nauseous, penetrating odour is exhaled. It may present these features from the outset, extending gradually from a restricted area, without any attempt at healing; or it may develop out of what was a pemphigus with tense bullæ by a process of degeneration. As the disease progresses, the hair falls off, the skin of the face contracts, causing ectropion, and emaciation occurs.

Diagnosis.—In diagnosing pemphigus, it must be borne in mind that blebs are met with under many circumstances as accidental rather than essential features. Thus they are seen in erythema multiforme, in urticaria, dermatitis herpetiformis, erysipelas, syphilis, and cheiropompholyx, in scabies, from the ingestion of certain drugs, as iodide of potassium, and from the application externally of irritants, as cantharides, the mineral acids, and mezereon, used either purposely or accidentally. The syphilide which simulates the pemphigus of the infant is chiefly localised on the palms and soles, and is accompanied by other symptoms of inherited disease.

Treatment.—Arsenic is the most valuable remedy we possess in chronic pemphigus of the ordinary type. Beginning with small doses, it should be gradually increased till some effect on the disease is produced, or indications that a sufficiently high dose has been reached are manifested. Other medicaments are quinine in full doses, and iron. The diet should be nourishing, but plain. Locally, calamine lotion with boric acid, or the following modified from one recommended by Unna, are grateful, and serve to prevent the formation of fresh bullæ—℞ Liq. calcis, olei olivæ, āā uncias tres : cretæ præp., zinci oxidi, āā semi-unciam ; acidi salicylici, 1 drm.—*M.* In cases where there were few bullæ, nothing has suited better in my experience than the continuous application of poultices of cold starch, containing boric acid, locally, and arsenic internally. Dusting powders, as a 3 per cent.

of salicylic acid, or a 10 per cent. of boric acid in talc, are useful, especially in pemphigus foliaceus. In it the continuous bath has been found valuable by Hebra and Kaposi. Hutchinson has found the liq. opii sed., 3 to 10 minims thrice a day, curative in mild examples of pemphigus vegetans, and to exert a controlling influence on the severe. A severe instance which came under my notice recovered under large doses of cinchona with arsenic, continued for months, with the lotion formulated above, locally.

DERMATITIS HERPETIFORMIS.

A chronic, rarely an acute, neurosis of the skin, not markedly interfering with the general health. This causes a more or less universal grouped eruption, erythemato-bullous in the main, associated with itching and burning sensations, and regularly recurring after intervals of complete or comparative immunity. Bazin recognised it, and pretty accurately described it, but failed to attract general notice. Tilbury Fox was acquainted with it, but it is to Duhring that we are indebted for bringing it prominently and permanently forward. Others, as Brocq and Unna, have further elaborated his picture.

Pathology.—As regards the pathology, two opinions are held—one, that it is a tropho-neurosis. In its favour are the paræsthesiæ, the symmetry of the lesions, and the recognised possibility of the evolution of vesicles in association with nervous affections. The other that it is due to an auto-intoxication by leucomaines, those alkaloids produced, according to Gautier, by the metabolism of the albuminous tissues independently of bacterial agency. Gaston has found renal sclerosis. Leredde holds that a toxic substance eliminated in health in the urine, is no longer so. This toxic substance acts, on the one hand, on the nervous system, giving rise to the itching, etc. On the other hand, it is present in the blood and fluid of the bullæ, thus determining the eruption. In support of this view, the administration of iodide of potass in dermatitis herpetiformis at once intensifies the eruption. It is now accepted that the bullous form of iodic eruption which much resembles it is specially apt to appear when it is given in cases of renal inadequacy, or nephritis. The diminution of vascular tension in pregnancy, owing to the hydræmic condition, favours its occurrence then. Nervous shock has started an outbreak in some instances.

Histology.—The histology has been investigated by Elliot, myself, and Unna. The vesicles are chambered and form in the substance of the rete, which is œdematous. The loculi are bounded by septa composed of somewhat degenerated rete cells, but are further subdivided by fine structureless dissepiments. Beneath the vesicles the limit was indistinct, the deepest rete cells being connected directly with the cutis by filamentous prolongations. There is no marked leucocytosis. Leredde and others have shown that there is simultaneous presence of eosinophile cells in the blood and serum of the bullæ, and Morris that the eosinophilia increases as each attack approaches its acme. Elliot found peripheral parenchymatous neuritis.

Symptoms.—The polymorphic nature of the eruption is one of its special characters. Erythematous blotches or flat papules are to be seen at some, usually an early stage, and make up the bulk of the lesions, but vesicles, bullæ, or pustules are met with also. The lesions are grouped, herpetiform, not isolated. The crusts or scabs which succeed the vesicles are hard and angular. In distribution, a partiality is evinced for the neighbourhood of the articulations, and for covered parts. Blotches or stains, pigmentation or white spots, are present on the subsidence of the more acute symptoms. Itching, often intense and aggravated at intervals, is complained of. When a new crop of lesions is about to appear, it asserts itself, calming down with their full evolution. Puncturing the vesicles affords relief. The disease runs an essentially chronic course, tending to recur or relapse. It is remarkable how well the health is maintained, when the extent of surface involved and the restlessness due to the pruritus are considered. It may occur at all ages, may last many years, and while it usually terminates spontaneously, it may end fatally. The name *herpes gestationis* has been given to it when, as it sometimes does, it appears during pregnancy or after delivery. Once having appeared, it is apt to recur in successive pregnancies, though one or more may be missed out. Opinions differ as to whether the rare and fatal disease, originally described as *impetigo herpetiformis*, should be dissociated from dermatitis herpetiformis. In it, small yellowish superficial pustules appear, which begin as such and continue so. These are arranged in groups or circles on an erythematous base. They dry up into greenish or brownish crusts, covering an excoriated surface, healing without cicatrix. Round these fresh groups or rings of pustules develop, so that wide areas are involved. Each outburst is preceded by rigors, fever, and systemic disturbance. The disease is pustular throughout, and its occurrence is almost exclusively limited to pregnant women.

Diagnosis.—It must be distinguished from erythema multiforme. The intense itching, the mode of evolution, the arrangement of the lesions, and the prolonged duration, all aid in this. It is most apt to be confused with scabies, but there is entire absence of communicability.

Treatment.—Arsenic occupies the first rank; it possesses a powerful inhibitory effect, but can hardly be called absolutely curative, since the complaint reappears on its discontinuance. The best form is the liq. arsenici hydrochloricus, which, in the anæmic, can be prescribed with the tinct. ferri perchloridi. Locally, baths containing liq. potassæ permanganatis give relief. Ichthyol, diluted with three parts of water and painted on, is one of the most serviceable applications. Sulphur ointment, 1 to 3 drms. to the ounce, rubbed pretty firmly in, is also a good remedy. Bearing in mind the probable pathology, attention should be paid to the kidneys; elimination by them should be promoted, in particular by the free exhibition of milk.

HYDROA VACCINIFORME.

Syn., Hydroa Æstivale.

An eruption recurring during the warmer two-thirds of the year, and in the period of childhood or adolescence, generally vesicular, and apt to leave sores. Bazin described this disease, but only one phase, hence his account of a rare complaint failed to attract attention. Hutchinson has under various names, particularly as a recurring summer eruption, referred to an allied if milder form.

Symptoms.—In its more severe variety, it is almost exclusively confined to males, though it has been seen in girls. It commences, as a rule, in early childhood, and recurs throughout youth, apparently ceasing with the completion of growth. In some instances there seem to have been attacks in winter, but in most the recurrences take place between February and October. Exposure to the rays of the sun, to the air on the sea, or to the reflection from water, promptly evokes an outbreak, and the acme of the annual seizures is always in the hot season. The ears are specially affected, the face, neck, and backs of the hands, the legs if exposed. Sensations of burning, tension, or pain, rather than of itching, precede each attack. Red erythematous blotches come out, and on these flat vesicles form, from a pea to a sixpence in diameter. The centre of the blister dries and assumes a reddish brown tint, the margins remaining vesicular. Or the blisters rupture, their contents becoming thick, friable crusts. The duration of an outbreak is about three weeks, then a fresh crop appears at once or after an interval. As a result of these repeated attacks, the ears may be reduced to little more than gristle covered with a thin cicatrix, and pitted scars, with some pigmentation, persist. The recurrences become fewer, and the attacks themselves milder towards puberty, and cease about twenty. In the slighter variety the eruption is sometimes erythematous, sometimes papular or abortively pustular. It may be more widely distributed over the body, leaving the palms, soles, and flexures free. It is associated with itching, occurs nearly exclusively during the warmer months, and leaves small scars. M'Call Anderson has related two cases in which hæmatoporphyrin was persistently present in the urine in each annual attack. There is a certain though ill-defined relationship with xeroderma pigmentosum. Severe sunburn, or long-continued exposure to Alpine snows, have in adults produced a similar idiosyncratic sensitiveness to a portion of the solar rays.

Treatment.—Since the condition of skin and system which predisposes to this is wholly unknown, our treatment must consist in protecting the skin by painting it with calamine or lead lotion, coloured brown with umber. Wearing a brown veil guards the skin considerably. All irritants are to be avoided, and only the blandest soaps employed.

PSORIASIS.

A disease characterised by masses of dry, white, shining scales, forming small punctiform elevations, or more extensive patches, round, oval, or annular, resting on a well-defined reddened base, which bleed readily from punctate apertures. A disease so common and with such distinctive characters could not fail to be well known from the earliest times, and Hippocrates, Galen, and Aëtius were acquainted with it. Willan formulated the definition nearly as given above, but he erred in calling the annular variety lepra, a term now properly reserved for leprosy.

Etiology and pathology.—The most recent observations seem to show that the earliest morbid change is in the upper layers of the epidermis, a too rapid and at the same time imperfect cornification of the corneous layer, — parakeratosis. Succeeding this occurs, almost simultaneously, epithelial growth, acanthosis, shown by prolongation downwards of the interpapillary cones or ridges, with corresponding elongation of the papillæ, and vascular dilatation. The tissues are œdematous, there is some leucocytic emigration near the vessels. The appearances favour the parasitic origin of psoriasis, but this has not yet been proved. Even in the healthy psoriasis skin there exists a peculiar predisposition to the eruption, so that superficial irritation, friction, a blister, the rash of scarlet fever, may serve to produce or precipitate an outbreak. The silvery hue of the scales is due to the inclusion of minute particles of air within and between the loose, imperfect, spongy masses. It is often hereditary, certainly not so always, yet it is rare for more than two members of even a large family to be affected with it. It commonly develops in youth, but may show itself for the first time at any age. In France, but sometimes in Britain also, arthritic deformities are met in association with it. Aggravations of psoriasis are apt to occur in spring and autumn, or during lactation, while the eruption fades during a severe illness, and reappears with convalescence. School life intensifies the complaint in children, and renders treatment less satisfactory.

Symptoms.—The first manifestations of psoriasis consist of minute scaly spots, each with a red margin. These are seated on the limbs, generally on their extensor aspects, particularly on or about the elbow, or under the patella, or on the scalp. With the exception of the last-named locality, they chiefly affect covered parts, rarely appearing on the face or the hands. The spots gradually or rapidly increase in size, but not all equally, though in the larger and smaller alike the circular shape is preserved. The scales which cover them become thicker, the mass having a silvery white or yellowish white tint, conveying the impression of a drop of mortar adhering to a patch of thickened, reddened skin. Beyond the scale there is always a red margin, so long as the disease is extending in area; when the process is quiescent, the hyperæmic zone may disappear, and be replaced by a pale one. Though the patches are essentially dry, if the scales are dislodged, a smooth, red, shining disc is disclosed, which may be very slightly moist. If this is scratched with the nail, tiny drops of blood are seen to ooze from the apices of the papillæ. Itching is seldom complained of except in cases where numerous spots develop quickly.

The patch may, however, enlarge at its periphery while the centre heals. Should the circle so produced encounter a similar one, retrogression takes place at the points of contact, and gyrate lines are formed. On the head, if the hair is plentiful,—and psoriasis *per se* is seldom a cause of baldness,—the masses of scales are often better felt than seen. From the abundance of sebaceous secretion and accumulated dust, their colour is yellowish or brown. The disease sometimes creeps on to the forehead as a prominent red line. In those who are bald or with thin hair the condition is rather that of dry, red, slightly scaly patches. On the scrotum, there is redness, induration, and cracking, giving rise to pain. Though fairly common on the backs of the hands, it but seldom affects the palms or soles. When it does occur there, small areas of thickening arise, which, as Crocker well phrases it, produce whitish worm-eaten spots. At the same time there may be dryness over the greater part of the surface, with the formation of scales. Occasionally a further development ensues; a degree of papillary and epidermic hypertrophy arises, a warty state, so that the soles present the aspect of a coarse yellowish white plush. Should this persist, an epitheliomatous degeneration may occur, as in a case recorded by White. The nails too are sometimes affected. The disease starting at one corner, may creep down the side; or a commencement may be made at the matrix, or in the bed, so that the nail becomes opaque, thickened, discoloured, and furrowed transversely. The epidermic accumulation may even separate it from its attachment.

Diagnosis.—The diagnosis is seldom a difficult matter. The various forms of seborrhœic dermatitis do closely resemble it. In them, however, the scales do not reach the thickness they do in psoriasis. In the scaliness which accompanies disseminate ringworm of the scalp, the microscope will at once clear up the difficulty. A dry form of eczema disposed in patches may rather nearly approximate to a psoriasis in the anæmic or very fair. The same may be said of the early scaly syphiloderm. In eczema there is usually the history of moisture at some stage, in the syphilide the examination of the throat and glandular system will help to discriminate. The patches of old-standing lichen planus are sometimes mistaken for psoriasis, but it is generally possible to discover some of the flat topped, shining, dull crimson red, angular papules at the margin. Pigmentation sometimes follows psoriasis, especially if treated by prolonged courses of arsenic. This must not be confused with that seen in syphilis or lichen planus.

Prognosis.—The prognosis of psoriasis is good so far as the removal of the existing eruption is concerned, but we are powerless to effect a certain and lasting cure. Still many cases, thoroughly treated, may continue for an indefinite period free from relapse. There is a tendency for psoriasis in some instances to change its type, to pass as a steadily advancing wave into general exfoliative dermatitis. In chronic alcoholics, too, the patches are apt to be large, and the scales which invest them to be brownish, but with the discontinuance of the stimulant, and under appropriate remedies, the disease often clears up rapidly.

Treatment.—In the treatment of psoriasis it is of great importance to distinguish between those cases in which the disease is spreading actively, and those in which progress has ceased, and a quiescent condition or one of natural retrogression has been reached. We do not possess any very efficient methods of arresting its advance. Something may be done by the administration of prolonged simple warm baths, perhaps also by the exhibition of salicin or the natural salicylates, as suggested by Crocker. If the disease is no longer progressive, and is widely spread, the employment of baths kept between 95° or 99°, medicated by the addition of carbonate of potass or potassa sulphurata, for not less than an hour nightly, and followed by the inunction of vaseline, if a somewhat tedious is a safe and satisfactory mode of removing the eruption. When the patches are few in number and thoroughly chronic, washing them with friction with Hebra's sp. saponis kalinus and warm water, to remove the scales, and then the application of chrysarobin, gives the most rapid results. Chrysarobin may be employed in ointment form, 10 to 30 grs. to the ounce, according to individual susceptibility, or as a 10 per cent. solution in liq. guttaperchæ. Since it is liable to induce a troublesome conjunctivitis, if used for the head or face, a 5 per cent. pyrogallic acid ointment, containing also a little salicylic acid and ichthyol, should be substituted for these regions. Both remedies stain the linen indelibly. Arsenic has long enjoyed an exaggerated reputation in the treatment of psoriasis. It acts best in first attacks, and is only suitable for thoroughly chronic cases. The disease may be kept somewhat in check by its administration, but is never wholly removed by its means alone. There is some evidence in support of the view that the epitheliomatous degeneration alluded to as occurring on the palms and soles, may be traced to its too prolonged use. It may be given as arsenious acid, $\frac{1}{50}$ to $\frac{1}{20}$ gr. thrice a day in pill, or as the liq. arsenici hydrochloricus in 4- to 8-minim doses, well diluted, and immediately after meals. In psoriasis of the nails it is our best drug. Thyroid tabloids may be tried in similar cases, but the action is even more uncertain than arsenic. Small doses must be used to commence with, and cautiously increased, the pulse being watched, and other symptoms of thyroidism looked for. It must not be given in alcoholic cases, or in the aged with weak hearts, and the patient should either be in bed, or under careful supervision. The diet in psoriasis needs only attention to ordinary rules.

PITYRIASIS ROSEA.

This is a widely spread eruption, characterised by pale red or fawn-coloured, slightly scaly patches or discs. This rather uncommon ailment was first described by Gibert, afterwards by Hardy, Duhring, and Brocq. According to the latter author, it always commences by the appearance of a single patch, which he calls the primitive one, and which may be seated anywhere on the trunk or neck, sometimes on the arm. This patch is oval or round, the margins are a tolerably bright rose colour, slightly elevated above the surface, and bearing a few fine scales, arranged sometimes like a collarette. The centre is a brownish, rather faded red. In size it varies from a quarter of an inch upwards. In course of a few days many more spots, similar to the first, appear. These are found on the neck, sides of the thorax, abdomen, limbs, but less frequently on exposed parts, progress usually being from above downwards. When fully developed, there are innumerable patches of a reddish fawn colour, or simply fawn, the rose margin having faded. These are very slightly scaly, not much elevated, and itch little. It is most frequent in females. Apart from micrococci, not apparently distinctive,—indeed, Unna states that any readily recognisable organism is absent,—there is no proof of a parasitic cause. It has distinctive microscopic and clinical features, which separate it from seborrhœic dermatitis. In that special form of the latter which is localised to the centre of the chest and back, the margin of the patches is linear, crescentic, and punctate, the red spots corresponding to the follicular orifices; there is a yellowish tinge, the skin is greasy, and generally thick flannels are habitually worn. From a macular syphilide the absence of any constitutional symptoms will serve to discriminate. It tends to fade spontaneously in a month or two. Warm baths of liq. potassæ permanganatis, 1 oz. in 30 gallons, followed by vaseline inunction, caused its rapid disappearance in some cases. But when this failed, it has yielded to washing with resorcin and salicylic soap daily, combined with the same application.

PITYRIASIS RUBRA PILARIS.

A symmetrical extensively distributed disease, characterised by the production of acuminate red papules, with a broken hair in their centre, a general redness of the surface, and the formation of a white limelike scaliness over large tracts of skin. Claudius Tarral, an Englishman, seems to have communicated the first recorded case to Rayer in 1828; but Devergie, Richaud, Brocq, but more especially Besnier, in an exhaustive monograph, have clearly described the features of the complaint. The disease is a rare one; but recently examples have come under the care of Liddell of Harrogate, Morton of Glasgow, West, Abraham, and myself, while various observers have encountered instances abroad. The disease pathologically is a diffuse hyperkeratosis, which is progressive in character, and finds its most marked expression at the hair follicles.

Symptoms.—It usually commences by the formation of scaly patches on the palms and soles, and by a seborrhœic condition of the scalp. Or it may appear on the face as a dry bran-like adherent coating. Or again the peculiar papules may primarily show themselves on the limbs or trunk. These are about the size of a pinhead, are acuminate, red or brownish, hard, and dry; each has a broken-off hair in its centre, which is surrounded by a horny collar, dipping into the follicle. These are seen very characteristically on the dorsal aspect of the phalanges of the fingers and thumb, but also on the limbs where most hairy. They are met with likewise on the trunk. As they increase in number they are aggregated into more or less extensive areas. These appear yellowish or reddish, feel thick and immobile, and are covered with branny scales, or with an investment like plaster. The lines become coarse, the aspect somewhat ichthyotic. Papules may be seen at the margins, or these may be wholly or entirely masked; but if by means of oil packing this superincumbent epidermic accumulation has been removed, dull, brownish red, smooth papules, not unlike those of lichen planus, are disclosed. The integument of the face is stretched and reddened, giving rise to ectropion. The nails are usually attacked, are greyish and striated longitudinally. The general health is little affected; itching, if present at all, is commonly moderate. It may begin at any age, and last indefinitely, partially disappearing at times.

Diagnosis and treatment.—It has been confounded with eczema, lichen planus, lichen pilaris, ichthyosis, and sometimes with psoriasis, to which, indeed, it seems closely related. In treatment, arsenic seems injurious. Benefit resulted apparently in West's case from thyroid tabloids; but local remedies, baths, oiling, and friction, were simultaneously used. In Morton's, thyroid did not check its spread during a relapse. Local emollients and pilocarpine are specially indicated.

LICHEN PLANUS.

A disease characterised by the eruption of flat, shining, pale, lilac, or crimson red papules, and of patches formed by the coalescence of these, multiform within certain limits, more commonly chronic and circumscribed than acute, and nearly universal. It was first distinctly isolated by Sir Erasmus Wilson. Hebra also described it, but he included an acuminate form, which, however, is probably pityriasis rubra pilaris.

Etiology.—Only certain facts can be stated as to the etiology. It is more common in the better than in the lower ranks of life. Some affected with it have been subjected to worry, others are neurotic, while a gouty element may be traced occasionally. It may occur at all ages. Colcott Fox has recorded cases apparently of lichen in infants; it is not infrequent in stout women over forty. There is no constant disorder of digestion associated with it. Sleeplessness is complained of by some. It is sometimes of very protracted duration; but is finally recovered from in most cases, though it may recur more than once.

Pathology.—The microscopic appearances are characteristic, and have been very fully described by Unna. There is an initial hyperplasia of the prickle layer, to which is added hyperkeratosis with delayed cornification.

The proliferation of the prickle layer diminishes, while the tendency to the formation of a specially firmly connected horny layer persists. The prickle layer undergoes widespread colloid degeneration, with permanent intercellular œdema. In the cutis the blood vessels are dilated, and there is a dense infiltration of cells, generally regarded as leucocytes, but this explanation is disputed by Unna. The livid, bluish red colour is due to the thick cellular layer, lying like a dense opaque medium over the dilated capillaries. The waxlike glance arises from the stretching of the horny layer from the abnormal pressure. There is a peculiar firmness and power of resistance inherent in the epithelium of the papule, in consequence of which it remains dry throughout.

Symptoms.—The disease is oftenest seen in the chronic form, which may be taken as the type. It commences usually on the limbs, specially on the front of the wrist or inner side of the knee. The papules which appear are rather angular or polygonal than round, and vary in size from a minute point to a hemp-seed. Each rises abruptly from the surface, and has a shining, waxy, flattened, rarely hemispherical summit, in the centre of which in some cases there is a tiny depression. Individually they are discrete or in lines. Their colour may differ little from that of the skin, but is more usually a deep crimson, or lilac red. Some, often severe itching accompanies their evolution, and persists. The angular papule does not increase much if any, but the round one may attain the size of a pea. New papules form near the old, or arise in fresh situations, anywhere, indeed, except on the face. As they become crowded together, the *patch* is produced. This is irregular in shape, though sometimes circular, well-defined, and slightly scaly. On the front of the legs, the inner side of the knee and thigh, the thickening and elevation of the patch may reach considerable proportions, the surface becomes rough and warty, being covered with greyish, pretty firmly adherent scales, while round is a more or less extensive ring of pigment. This has been called *lichen verrucosus.* The disease advances slowly in the chronic variety, and may remain unaltered for years. During involution the papules assume an even darker tint, till merely a brown staining, which fades very leisurely, is all.

Occasionally lichen planus develops rapidly, and invades in a comparatively short period the greater part of the covered regions. The papules present the same features, but are generally a more decided crimson red. The patches are numerous, less sharply defined, bear small branny scales, itching is severe enough to prevent sleep, and mottled pigmentation succeeds.

Sometimes the palms are affected; there are dry patches from which the horny layer peels off, and glassy, horny papules, yellowish in hue. On the tongue and buccal mucous membrane there are spots and lines of a whitish colour rather than papules.

Diagnosis and treatment.—Eczema in its dry and papular form may resemble lichen planus, but the papules are more pointed, and there is an absence of the burnish, while there is polymorphism. This latter symptom too will serve to assist in distinguishing from the small papular syphilide, with which there are also constitutional symptoms. In treatment, arsenic takes the first rank; but to do good it must be given in full doses, and continued for a long time. One of the best forms is the liq. arsenici hydrochloricus, with which may be combined dilute hydrochloric acid, and if there is much itching, small doses of the liq. strychninæ hydrochloratis. When arsenic fails, the tartrate of antimony,

$\frac{1}{8}$ to $\frac{1}{16}$ gr. three times a day, is sometimes successful. Locally baths of potassa sulphurata at blood heat, followed by vaseline inunction, are soothing where itching annoys, and an ointment containing chloral camphor is valuable for the same purpose.

PARAKERATOSIS VARIEGATA.

This is the appellation which Unna has attached to a peculiar, rare, chronic skin disease, allied to lichen, but presenting some marked distinctive features. Three undoubted instances have come under my care, and probably two others, though milder. It commences in adult life, about the clavicles or on the legs, by the appearance of minute crimson red spots or flat papules. These have a certain degree of smoothness, but scarcely the burnished glance of lichen planus. These increase in number linearly, then coalesce so as to give rise to a species of meshwork enclosing paler areas. It extends gradually over the trunk and limbs, but does not affect the hands or feet. On the face and head there is rather an erythematous or plum-coloured redness than discrete spots. A slight degree of desquamation is seen here and there. There are no subjective sensations, though the skin is sensitive to cold, and the health is maintained. The redness extends, producing a darker and paler crimson mottling. The hairs fall out, and there is an atrophic thinning of the integument. In one of my cases, after many years, small soft tumours, resembling those of mycosis fungoides, formed in places, burst, exuded a watery serosity, then became absorbed. According to Unna—and sections from one of my cases agree with his—the morbid appearances are limited to the epidermis and papillary body. There is some dilatation of the vessels, œdema of the tissue, and distension of the lymph spaces, thus causing tension of the corneous layer. The nature of the complaint has not been made out. It is possible, as was suggested by Malcolm Morris, that it may prove to be the premycosic stage of mycosis fungoides. But a case apparently identical, spontaneously disappeared. No treatment of any kind had the least influence.

GENERAL EXFOLIATIVE DERMATITIS.

An inflammatory condition affecting the entire surface, characterised by vivid redness with copious branny or flaky desquamation. Devergie described this; Hebra somewhat limited the conception, and called it pityriasis rubra; since then Brocq, Buchanan Baxter, Stephen Mackenzie, Savill, Elliot, Jadassohn, and others have further contributed to our knowledge.

Etiology.—The cause seems variable. Irritant applications, chrysarobin and arsenic in psoriasis, arnica in some, a tuberculous element according to Jadassohn, have all been blamed. A vasomotor disturbance or some spinal affection may underlie it. The epidemic form chiefly seen in workhouses or hospitals, though similar cases occur sporadically, appears to be contagious; a microbe in some respects like the *Staphylococcus albus* was found by Savill and Risien Russell.

Symptoms.—The three features which come prominently forward, are the universal distribution, the redness, and the desquamation. The redness varies from a vivid hue to a duller tint. It fades on pressure, a yellowish tinge remaining. Sometimes no, at others a degree of thickening is perceptible on pinching up. The scales are largest on the back and extremities. They may be thin, dry flakes, attached in part, in part free. Occasionally, or in some forms, there is a slight amount of moisture. On the face they are branny, while on the scalp they become matted together into a species of crust. The palms and soles may escape, or be involved late in the disease. Should it last long the nails and hair participate, the nails becoming discoloured, split up, or are shed; the hair acquires a dull and withered appearance, and gets thin.

It many cases it is surprising how little the health suffers from such a widespread and protracted disorder. Feverish symptoms accompany it in some instances. A very common complaint is made of chilliness or great sensitiveness to cold, and the respiratory and renal functions are apt to be disturbed. The extension and the duration are alike variable. In chronic cases there are remissions and exacerbations, and even after cure has resulted, it may relapse. In infants and in the epidemic type described particularly by Savill, the mortality is considerable. Though it may be primary, it more frequently develops out of, or directly succeeds some other affection. Thus some cases of psoriasis pass into it, or it may be the sequence of an eczema, more rarely of a lichen planus. Pemphigus foliaceus has been rightly regarded as a moist form, and there is a moist variety of the epidemic type. Men suffer from it more often than women.

Diagnosis.—The diagnosis can seldom give rise to difficulty. It may sometimes be a little hard to decide whether a case should be described as an extensive eczema or an exfoliative dermatitis. Erythemata followed by desquamation, and dubbed scarlatiniform, are rather like it. In such the separation of the epidermis as branny dust, and the superficial nature of the process, would distinguish.

Treatment.—The remedy which in the dry forms at least gives best results, is the administration of tartrate of antimony in small doses for a considerable time. Quinine or cinchona is also useful. Locally, bran or starch baths are soothing, followed by vaseline inunction. Or calamine lotion with boric acid may be freely painted on, or the skin dusted with a boric acid and talc powder, or with emol kelect. With the chilly feeling already mentioned, there is also a sensation of burning or itching, so that irritation or exposure must be sedulously avoided. The food should be unstimulating, and the function of the kidneys should be promoted by diuretics, or at least draughts of water between meals.

DERMATITIS.

This is a generic term including a number of inflammatory states of the skin, the qualifying adjective indicating the cause, rather than the form and appearance.

Dermatitis Traumatica.

Such are eruptions due to mechanical causes, as pressure, friction, but especially scratching, occasioning thickening, pigmentation, and abrasions.

Dermatitis Calorica.

The extremes of heat and cold, if their action is intense or prolonged, equally give rise to inflammation. Death of the part may result from cold by deprivation of blood, or excessive sudden reaction. The sun's rays cause congestion or vesication, but destruction only follows contact with heated liquids or solids.

DERMATITIS VENENATA.

This includes various inflammations set up by external irritants as well as trade eruptions produced by substances only noxious to particular individuals or at certain times. Aniline dyes, the red most commonly, but also other hues, probably because they contain arsenic in some cases, through the medium of the clothes coloured with them, cause a reddish papular condition with itching, which may spread beyond the part directly in contact. Arnica is a fruitful source of an erysipelatous-like irritable eruption, when applied to sprains, for which it is a popular, though useless and harmful remedy. Bichromate of potass in the form of strong solutions, causes papules and pustules, and later ulcers and sloughs. Chrome mordants used for fixing colours are occasionally an unsuspected cause of ulcerations about the nails in those sewing the cloth, or of itching and sores on various parts of the body in those wearing it. The hairs of the primula obconica set up, in some of those who handle the plant, an itching eczematous-like eruption. Those occupied in extracting the alkaloids of opium sometimes suffer from an irritable erythematous or scaly eruption on the arms, face, and neck. The treatment of such conditions consists in removal of the cause, and the application of soothing lotions, as calamine or weak lead ones, or of dusting powders of emol keleet or boric talc.

DERMATITIS FICTA.

Hysterical women, mendicants, and impostors are in the habit of employing various irritants for some morbid reason, or to provoke sympathy. The operators so well conceal their methods, that detection is not easy, and in the case of the hysterical is apt to be resented by relatives. The lesions so produced are not very like any known disease, are usually on accessible parts, and principally on the left side in right-handed persons. Carbolic and nitric acids are those chiefly applied, often so severely as to give rise to gangrenous areas, which may leave scarring. The services of a trustworthy nurse may be required to unravel the mystery satisfactorily.

DERMATITIS MEDICAMENTOSA.

The literature of drug eruptions is an ever-increasing one, since so many of the new synthetic remedies, and some of the animal extracts lately introduced, have added to an already large list. The work of Morrow, edited for the New Sydenham Society by Colcott Fox, will be found well up to date. It is impossible to do more than briefly allude here to some of the more prominent. Idiosyncrasy plays an important part in their production, or there is renal inadequacy, or a cardiac ailment interferes with the due elimination. Iodine and bromine cause distinctive and generally typical eruptions, those following the administration of other substances are less plainly indicative of the source. As to the manner in which the eruption is induced, one may conceive that the influence is reflex through the vaso-motor system in cases where the drug is introduced into the stomach, or applied to a mucous canal; or if the drug or its product enters the circulation, it may act directly on the peripheral nerves or the integument, or may cause irritation while being eliminated by the cutaneous glands. Behrend holds that it is not the drug itself, but some toxine produced by its absorption or excretion which occasions the eruption. It has been suggested that certain drugs have affinities for particular elements of the tissues, but the absolute proof has not yet been furnished.

Antifebrin.—If the dose is large or administration long continued, cyanosis is produced.

Antipyrine.—Exceptionally after a single dose, ordinarily only after a course lasting several days, a rash may appear. The usual form resembles that of measles, but commences on limbs, and is little pronounced on face. Erythematous patches and purpuric spots have also been seen. It is frequently attended with profuse sweating, and more or less itching. Desquamation may follow. Bullæ limited in amount or widespread occur, apparently from large doses.

Arsenic.—An erythematous extensively distributed eruption of an intense or light red may appear. Or papules at first in scattered groups, later as patches. Œdema of the eyelids and conjunctivitis are not uncommon, after prolonged exhibition. Tenderness of the palms and soles, with hyperidrosis, occasionally ensues in similar circumstances, thickening of the epidermis round the sweat pores succeeds, then diffuse hyperkeratosis, which may pass into epithelioma. Herpes zoster has been observed during a course, often a first, and vesicular eruptions somewhat like eczema. Pustules with subsequent ulcers may arise. But the most constant sequel is brownish discoloration on the face and other regions. It disappears by desquamation in about a month after discontinuance.

Belladonna.—A bright diffuse redness, transient in character, commonly confined to the face and neck, but sometimes extending over the chest, or even universal, and very like the rash of scarlet fever, may follow its external or internal use. Atropine drops applied to the conjunctiva, may cause a troublesome acute inflammation of the lids and cheek, not unlike erysipelas. A severe dermatitis may follow the use of the A.B.C. liniment.

Borax.—This, when given for a long period in the treatment of epilepsy, has caused a scaly eruption quite like psoriasis, or resembling eczema.

Chrysarobin, when applied in any form to the skin, is apt to produce a hyperæmia with purplish or prune-juice discoloration. Sometimes the eruption is erythematous or papular. Little black points, corresponding to a pilo-sebaceous follicle, are also seen. If used to the scalp or face, localities totally unsuited for it, or to the genitals, swelling and œdema and painful conjunctivitis may occur. It may even produce a pustular or furuncular eruption.

Bromides.—Any of the bromine preparations are liable to produce eruptions, which generally assume the pustular type, though erythematous, urticarial, and bullous have been met with. Bromic acne, as the discrete variety has been called, is frequent in persons taking continuous doses, as in epilepsy. As in acne, it affects by preference regions well supplied with sebaceous glands, is not limited to the face, chest, and back, but occurs on the limbs; and the papules, tubercles, and pustules are not preceded by comedones. The confluent form is more common in infants than in adults. In it, bright red, oval, convex elevations appear on the face and limbs, bearing on their tops closely set pustules. The swellings themselves are soft. A thick yellowish or dark scab forms, under which is a shallow ulcer, which on healing leaves a stain but rarely a scar. Crocker has remarked on the tendency these have to commence in cicatricial tissue. The eruption may not manifest itself till some time after the drug has been discontinued. Large doses are more prone to occasion it than small. It was thought that the lesions took their origin in the cutaneous glands, but it seems more probable that they are only involved secondarily, the primary seat being in or near the vessels. The indication for treatment is the withdrawal of the remedy, but as this may be undesirable, the conjunction of arsenic has proved in some instances a corrective, or, as Féré found in a case where fungations formed, beta naphthol and salicylate of bismuth given with a view of obtaining intestinal antisepsis, led to the cessation of the eruption. Crocker suggests salol in 5 gr. doses as a substitute.

Chloral.—Individual predisposition plays a large part in the liability to eruptions from the use of chloral. Hutchinson figures a case where a dull red patchy eruption appeared on the backs of the hands, shortly after a moderate dose had been taken, was accompanied by burning and itching, remained out a week, then slowly vanished. The erythematous type is the more common. It takes the form of a diffuse redness on the face, but as patches or dusky irregular red spots on the rest of the body. It is usually transient, and no subjective sensations or constitutional disturbance accompany it. Hot fluids, alcohol, or a full meal, render it more intense and generalised. In other cases the rash may be morbilliform, or hardly to be discriminated from scarlet fever, since pyrexia is present, and desquamation follows. Papular, vesicular, and urticarial types have been met with. The eruption has occasionally assumed a purpuric character.

Copaiba and cubebs.—Both may occasion eruptions, but the former is certainly the chief offender. The commonest form is that in which discrete, minute, erythematous points, apparently follicular, but tending to become grouped into patches, appear about the wrists, ankles, knees, hands and feet, chest and abdomen, though the rash may be universal. Itching, often very severe, accompanies it. Should the drug be discontinued, it rapidly wanes, a little branny desquamation occurring. There may be œdema, wheals may form, or vesicles appear. In some instances ecchymoses or petechiæ have been seen. As these drugs are so frequently prescribed in cases of gonorrhœa, the eruption may lead to the suspicion of syphilis, but besides the characters above mentioned the absence of constitutional symptoms, combined with a somewhat balsamic odour of the breath, will enable the diagnosis to be effected.

Iodine and iodides.—The eruptions which follow in some cases the ingestion of the preparations of iodine, are notable for the variety of the forms assumed, for their frequent occurrence after but small doses, for their prompt disappearance in general when further use is abandoned, and for their liability to appear in those who suffer from renal diseases or cardiac weakness, or who are at the time the subjects of bullous forms of erythema or dermatitis herpetiformis. Not in general of serious import, they may in exceptional circumstances be very grave, may even terminate fatally. Once induced, and having disappeared owing to the cessation of administration, a much smaller dose may serve to reproduce them. The papulo-pustular form is the one ordinarily met with. It appears on the face, front and back of chest and arms, though it may overstep these limits. The pustules, which develop from hard shotty papules, are rounded with a reddened and infiltrated base. The contents produce a crust, which may be followed by a cicatrix. An allied type pretty closely resembles a carbuncle, but the elements are numerous, reddish coppery in colour, and on being incised exude only blood. Iodic purpura has occurred shortly after the use of the medicine has been commenced, and from small doses in many cases. It appears more particularly on the legs. A peculiar and dangerous variety is the bullous, which indicates a special idiosyncrasy, and calls for immediate and final disuse. It has been seen after very small doses and after large ones, and even in an instance recorded by Dobie of Crieff, with fatal result in a man with Bright's disease, after painting with liniment of iodine. Other forms are the erythematous, the papular, and the urticarial. Mistakes have been made between some of the iodic eruptions and variola or syphilis.

Iodoform.—Dressings containing this have frequently produced a vivid erythema of so intense a type that erysipelas has been suspected. Considerable irritation accompanies it. In other cases it resembles rather a moist eczema. Vesicles and bullæ have also been seen. It is probable that the contained iodine is the cause, though the crystalline particles are more prone to provoke these effects than a fine powder.

Mercury.—Inunctions are apt to cause an eczematous-like eruption, and vaginal or intra-uterine injections of corrosive sublimate may be followed by punctiform erythematous rashes, scarlatina-like in appearance, by general exfoliative dermatitis, or by bullæ.

Morphine.—Opium and its preparations, as well as morphine, may produce a bright red erythematous or punctiform rash, accompanied with very severe pruritus. This does not apparently occur after its employment hypodermically. Repeated injections by those a prey to the morphine habit produce an appearance which may be puzzling. This may take the form of a symmetrical pustular eruption on the outer aspect of the thighs, but on close inspection it will be seen that the direction of the puncture marks is downwards on the right, but upwards on the left side. Those engaged in the process of extracting the alkaloids of opium are liable to eruptions sometimes erythematous, in others more like a chronic dry scaly eczema, with marked itching.

Quinine.—The external application of salts of quinine, as in form of pomades or of hair washes, or in the case of workers in quinine factories, has occasioned papular eruptions with itchiness. Its internal administration is also followed by eruptions of various kinds. These may appear in persons who had previously used it without such results. The most important type is the erythematous, which is generally of a vivid red, much like the rash of scarlet fever. It fades when the drug is given up, but is apt to be succeeded by desquamation. Urticarial and petechial forms are also met with, more rarely papular, vesicular, or bullous. Severe inflammation of the genitals has been noticed by different observers; in this, as on other parts, gangrene may ensue.

Rhus.—The dermatitis resulting from contact with the poison ivy or sumach is chiefly seen in the Western States of America. The hands are usually the parts first attacked, and by their agency the irritant is conveyed to other regions. The rash, according to White, is primarily vesicular, but erythema and œdema accompany or follow. It occupies from a few hours to several days to develop, with sensations of great heat and itching. Under treatment it may subside in from ten to fourteen days, but left alone lasts considerably longer. The lesions may be bullous or pustular. There is an opinion prevalent in the districts where it prevails, that an attack renders the person more liable to other cutaneous diseases. Soothing treatment is indicated, as calamine lotion with boric acid, or 1 drm. of the fluid extract of grindelia robusta in 6 oz. of water, painted on several times daily. The eruption may be obviated by thorough ablutions.

Tar, when applied externally, even in weak solution, in many cases is apt to produce an erythematous rash, extending beyond the seat of application, and accompanied by burning and itching. When rubbed in as an ointment, it causes plugging of the hair follicles, seen as a black point, followed by swelling and inflammation. Closely allied to this is the irritation due to petroleum and paraffin, met with in workers in shale. This consists of a dryness and harshness of the skin of the hands, arms, and legs, not unlike eczema. Or a folliculitis results, with epithelial hypertrophy of the hair-sheaths. If the employment is persisted in by those who exhibit this tendency, a further stage in which epitheliomatous growths develop is apt to supervene. The treatment consists in the application of poultices of starch containing boric acid, applied cold, till the irritation is subdued, afterwards vaseline with 2 per cent. of salicylic acid. If the work is resumed, the skin should be carefully freed from the oily matter by washing with a superfatted soap, and then powdered with boric acid, ten parts, in ninety parts of talc.

HYPERTROPHIC AFFECTIONS.

ICHTHYOSIS.

A DISEASE characterised by great dryness of the skin, locally or extensively, with the formation of scales, epidermic accumulations, or horny-like growths. The view of the disease is somewhat wider than that usually held, but it will be found to simplify the conception. A dry harsh skin, one in which there is an absence nearly everywhere of the slight unctuousness which is a normal feature in early adult life, is not uncommon, and is known as *xeroderma*. When the same condition is limited to the neighbourhood of the hair follicles in certain regions, it has been called *lichen* or *pityriasis pilaris*. If confined to the palms or soles, it has been named *tylosis*. When the epidermic accumulations become veritable spines or horny growths scattered over the surface, it is known as *ichthyosis hystrix*. The commencement is laid in intra-uterine life; a hereditary strain may often be traced, but the actual complaint may not be manifest till some time subsequent to birth.

Etiology and pathology.—The etiology and pathology have been made much clearer by the researches of Ohmann Dumesnil and Bowen into the subject of the human epitrichium. This is a temporary epithelial layer of the skin, which disappears at birth, when its functional necessity has ceased. It overlies the stratum corneum of the epidermis, from which it is anatomically and physiologically distinct. Dumesnil thinks that it is impervious to water, and may thus protect the integument of the fœtus from maceration by the liquor amnii, while it keeps the sebum or vernix caseosa to the surface. In the localities where the hair development is strongest, as the scalp and axilla, the separation of the epitrichium first takes place; and where the secretion of fat is most active, it is raised to the greatest degree. Should there be an arrest of development of the hairs and sebaceous glands, the raising of the epitrichium does not occur, and it becomes adherent to the stratum corneum. When this adhesion is generalised, it leads to the production of the various forms of ichthyosis. Its attachment on the palms and soles produces the great thickening of the epidermis in ichthyosis of these regions. Though congenital in its origin, yet most cases do not exhibit the peculiarities of the skin described till some months after birth, nor do these become very conspicuous till the second year. Occasionally some defects are seen at or soon after birth. The vernix caseosa may be abundant, but when washed the skin is a deep red, and has a varnish-like polish, being at the same time so stiff and tight that movement is scarcely possible; transverse and deep fissures form, dipping into the corium. These fissures divide the plates of thickened epidermis into irregular islands. The vitality is low, and most of the examples of "harlequin fœtus" die of chilling of the body and starvation.

Symptoms.—Adopting the comprehensive conception, the slightest manifestation is the heaping up of the epidermis round the hair follicles, giving rise to the formation of pin-head sized papules, either of the natural colour of the individual skin, or blackish from the accretion of dirt. The adjacent integument is normal or redder than normal, often very dry and scaly, feeling like shagreen leather. This is chiefly seen on the outer aspect of the upper arm or of the thighs. It is a source of annoyance to many young ladies.

In other cases the ichthyotic condition is strictly limited to an abnormal increase in thickness of the investment of the palms or soles, which may be accompanied with a degree of inflammation. These parts are covered with a dense yellowish horny plate of epidermis, so thick on the palms as to interfere very greatly with flexion; on the soles it may reach a thickness of fully a quarter of an inch. As a rule, this is dry, but there may be hyperidrosis, when it is sodden. It may be congenital and perhaps hereditary, or a very similar state may be induced by the long-continued use of arsenic. A somewhat similar acquired condition has been described by Brooke as *erythema keratodes,* and another by Besnier as *keratodermia erythematosa symmetrica.* The latter is distributed in patches, the former is uniform. There is great thickening of the epidermis, with a broadish ring of erythema as a margin, associated with a degree of heat and swelling.

In the diffuse form of ichthyosis the entire surface is seldom involved; the face, the flexures, the palms and soles are generally immune, while the rest of the body is affected in different degrees. On inspection, the skin looks dirty, and varies from a sallow to a grey or olive tinge. The surface feels dry, harsh, and rough, crossed by lines which may enclose lozenge-shaped spaces on which epidermic plates are piled. The lines are thinned portions, where the skin has yielded owing to the pressure and inextensible character of the plates. There is marked lichen pilaris, while perspiration is absent on the ichthyosed areas, the unaffected parts showing moisture in warm weather, or on exertion. Itching is not a conspicuous feature, unless when eczema is superadded, as is sometimes the case. The skin is apt to exhale a sickly, peculiar odour. Occasionally asthma coexists, though the attacks are less frequent if the condition is ameliorated by treatment. The hair is often dry, the scalp scaly, and the nails brittle. In most cases the legs exhibit the highest grade, the arms and trunk a slighter degree.

The hystrix form is never general, but may be extensive; it is less often symmetrical, may be not only unilateral but accurately limited by the median line. It may follow apparently the course of certain cutaneous nerves, forming transverse lines on the trunk, or longitudinal ones on the limbs. Yet it is doubtful if the warty growths, called, among other names, *nævus neuroticus unius lateris*, *papilloma neuroticum*, or *nævus verrucosus*, are rightly included under ichthyosis hystrix. In its most marked form there are warty, dark green, thickened masses or areas, or more or less scattered spiny prominences. These may exfoliate spontaneously, or may be caused to separate by maceration, when hypertrophic papillæ are disclosed.

Pathology.—The essential element in the morbid anatomy of ichthyosis is the hyperkeratosis. Cornification seems to be simplified, the prickle cells passing directly into horny ones, without the ordinary intermediate stages. The prickle layer itself is reduced in thickness, while the cells themselves are smaller than is customary, the lymph spaces being extremely narrow. The process extends into the neck of the pilo-sebaceous follicle, but, according to Unna, there is no alteration of the sweat pores. The coils, however, exhibit evidences of diminished activity. In ichthyosis hystrix the strata of horny cells dip deeply down between the elongated papillæ. There is in ichthyosis retention of the hypertrophied epidermis.

Diagnosis.—The diagnosis can occasion little difficulty, save perhaps in those cases where an extensive eczema complicates the disease. Except in the actually congenital instances, the prognosis is good as regards amelioration, and cure has resulted from early and persevering treatment.

Treatment.—This consists in softening the hyperkeratosis by warm alkaline baths, or by washing with a superfatted resorcin and salicylic soap. Thereafter a resorcin ointment of 3 to 5 or 10 per cent. should be applied. The excipient for the ointment may be lanoline thinned down with oil, whale oil being the most penetrating, or lard and lanoline. Subsequently the skin can be kept in good condition by washing with the above-mentioned soap and the application of thin glycerin of starch. Thyroid extract carefully used might assist the external measures. In ichthyosis hystrix the spinous formations can be temporarily removed by painting with a solution of salicylic acid in alcohol or collodium flexile. The harlequin fœtus should be kept in an oil-bath, or swathed in cloths saturated with oil, while it must be fed with a spoon or feeding cup, since it cannot suck.

VERRUCA.

An acquired excess of growth of the formative epidermic layers, in limited areas, with elongation of the papillæ, and increased cornification, of infectious or contagious nature, give rise to varieties of verruca.

VERRUCA VULGARIS.

This is the wart so frequently met with on the hands, chiefly in childhood or adolescence. There is at first an overgrowth of the deeper and superficial elements of the epidermis. This, while it flattens out many of the papillæ, causes a thinning and elongation of others, and there is produced a button-like body, often with a rough plush-like summit, enclosed by a horny ring, which advances up the side. It is dark in colour, partly from change in the keratine, but more from dirt. They are seldom numerous if the hands are cared for or protected, but may be plentiful in the neglected. Untreated, they frequently vanish mysteriously, it may be about puberty, but they may, on the contrary, persist.

A peculiar and painful variety of wart—the plantar—is sometimes met with on the sole, and especially at the distal end of the third metatarsal bone. Attention to this kind has been particularly directed by Dubreuilh. It is less prominent than those on the hand, has little black puncta, due to hæmorrhages, more frequent if it has been cut, and is generally surrounded by a hyperæmic, tender ring or halo. These are spontaneously painful, and are influenced by changes in weather. Some local irritant is often to blame for their causation, as seams, or pegs, or nails in the sole of the shoe. In seasons when the feet perspire much, and the sole is sodden, these are more apt to start the process.

Verruca Plana.

These are flat, not projecting much above the surface: though most common on the face and backs of the hands, they may occur elsewhere. They are soft, and are at times somewhat shiny, so that they resemble lichen planus. Pale yellow or fawn in colour, they do not indicate their presence by any severe itching. They have no tendency to spontaneous disappearance, and are very resistant to treatment.

Verruca Senilis.

These are met with in the elderly or aged, seldom under 40. They are seen on the back, arms, back of the hands, and upper part of the face. In the clean they differ little from the verruca plana, but in those neglectful of baths, there is a thickish greasy crust, yellow or approaching black, which can be scraped off, leaving a pinkish reticulated or granular surface, with bleeding points. They are associated with other symptoms of senile degeneration, but cause little annoyance save from appearance. Though in themselves innocent, they may, especially on the face, form the starting-place of an epithelioma, and this may, as Besnier has described and figured, eventuate in rodent ulcer of an aggravated type. Pollitzer has investigated their structure, and traced their connection with seborrhœa. According to him, the horny layer is slightly, the rete considerably, thickened, with epithelioid cells in the corium, combined with a peculiar infiltration with fat. Unna, again, regards them as allied to soft nævi.

Verruca Digitata.

These are less frequent, and are met with chiefly on the scalp. Their outline is irregular, the enlarged papillæ are long, ragged, and free. They are sessile, with a short stalk.

Verruca Filiformis.

These are long, narrow, thread-like, occurring on the neck or eyelids. They are possibly the atrophied portion of skin which has contained a small fibroma, and are not strictly warts at all.

Verruca Acuminata.

The so-called venereal wart, but, as indicated by myself and further demonstrated by Cathcart, they are contagious tumours, independent of gonorrhœa and syphilis. While they tend to be tufted and form fleshy excrescences of a cauliflower aspect, they may occur singly. They are pink, reddish, or even purplish. They are often bathed in a thin, acrid, puriform secretion, giving off a penetrating, disgusting odour. On the female genitals they are found in greatest luxuriance, but are met with on the glans, prepuce, at the anus, umbilicus, under the mammæ, or in the mouth. Untreated, they continue indefinitely. The medium of contagion may be organismal, but has not been isolated.

Treatment.—In the treatment of warts of all kinds, salicylic acid is invaluable, but is specially applicable to the ordinary and plantar wart. From 1 to 2 drms. are dissolved in an ounce of flexible collodion, or of equal parts of creasote and collodion. This is painted on the wart, and over it is placed a piece of galbanum plaster. The plaster is removed daily, and the soddened epidermic layers removed by pumice stone. Should this be too slow, the final result may be hastened by painting with a saturated solution of chromic acid, which has, however, the faculty of staining the wart black. This is peeled off each day. The plane and senile varieties are similarly treated. The acuminate, if small, yield to chromic acid; if large, require excision and the actual or thermo or electric cautery.

ACANTHOSIS NIGRICANS.

A disease having for its main characteristics general pigmentation of the skin and some of the mucous membranes, with warty growths, has been described under this name. Pollitzer and Janovsky recorded the two first cases, Morris fully reported one under his care, and apparently about a dozen have now been seen. There seems to occur first a bronzing of the skin, more particularly of the upper half of the body. Then on various regions, as the neck, axillæ, umbilicus, natal and inguinal folds, the hands and face, soft, papillary, or warty growths appear, not unlike plush or condylomata, but very dark or even coal-black. Warty growths, more or less stained, form also on mucous surfaces, as the palate or the vagina. Itching has been complained of, but as a rule there are no subjective sensations. The ages of those attacked varied from 35 to 62. In some there was more than a suspicion of internal malignant disease. A possible relationship to Addison's disease has been suggested. Unna, Clarke, and others have examined the skin and growths. There is increased pigmentation of the prickle layer, and staining of the horny one. The warts show an enormous increase in the prickle layer, with evidences of inflammation. The brown masses appear to be hæmatoidin, from minute intra-epidermal hæmorrhages. No theories of its causation or pathology, or suggestions for treatment, have been made.

CORNS—CALLOSITIES.

Corns are abnormal localised thickenings of the epidermis, with one or more conical projections downwards, occurring either over a bony prominence, as the second phalanx of the little toe, when a hard one is produced, or between two toes, when the soft is developed. Callosities are horny thickened patches on yielding portions of skin.

Besides the situations named, corns may arise on the fingers from pressure of the penholder, or even on the lip. They are due to intermittent pressure or friction, usually from ill-fitting boots. From the folding over of the smaller toes a corn forms either on the convexity, or on the extremity pointing downwards. The softness of the corn between the toes is owing to maceration. Both kinds may suppurate and give rise to painful ulcers, but apart from this they are also spontaneously liable to sharp stinging sensations. The nail-like projection digs into the cutis, rendering walking uneasy. Callosities are the result of an effort of nature to afford protection to a soft portion of skin, which is frequently subjected to pressure or friction. They are seen in many trades from the action of tools, or from occupation, as in boatmen. But they may appear without such evident cause.

Treatment.—In the treatment of corns particular attention must be paid to have suitable, easy, and broad boots, provided with stout soles. Over the corn itself a piece of plaster made by dropping melted emplastrum plumbi on white silk, is worn. This so far softens it that in a few days it can be picked off. But to prevent recurrence well-fitting boots must be worn, a different pair at least being put on on alternate days. Soft corns need the application of astringents, as spirit of camphor painted on at night, daily ablution, and separation of the surfaces with cotton-wool. Callosities generally yield to a suspension of the efficient cause. They need not be interfered with if due to occupation. If their removal is desiderated, it may be accomplished by painting on a 10 to 20 per cent. solution of salicylic acid in flexible collodion, which will in time allow the thickened mass to be peeled off.

HORNS.

These are rare, they are laminated or fibrillated, solid in consistence, and grey, greenish, or black. They are usually twisted or bent, and have a blunt or broken end, seldom pointed. If they are knocked off or drop off they leave a raw red, somewhat granular surface, on which another is apt to be reproduced. Essentially they are hypertrophied warts, and spring from the rete mucosum, or from the everted inner surface of a hair follicle. They are oftenest seen on the head, but may occur elsewhere. In treatment the whole base must be excised.

SCLERODERMA.

A diffuse or circumscribed induration, rigidity, or hidebound condition of the integument, which may be subacute or chronic. The first notice of this remarkable alteration in the skin seems to have been that by Cursio at Naples in 1752. Alibert and Startin recognised it, but termed it ichthyosis cornea. A notable paper on it was published by Addison, but by naming it "true keloid" he caused confusion. Fagge, however, placed it in its right position. Erasmus Wilson separated the circumscribed variety under the term "morphœa." More recently, Crocker and Hutchinson have added to our knowledge of its relationships.

Etiology.—Females are rather more prone to be affected than males, but no age is exempt. There seems to be an obscure association with rheumatism, and this may explain the part which some have thought that cold or exposure have to do in producing it. Elevated or nodular formations are found in some cases, resembling keloid. The fingers may be attacked, the *sclerodactylia* of Ball. Besides immobility, other changes of a vasomotor kind are then seen. These may be of the nature of local asphyxia, or whitlows, bullæ, ulcerations, and gangrene, so that one or more phalanges may be lost. These cases bear some resemblance to Morvan's disease and to *ainhum;* indeed, it is not impossible that the latter is really a form of scleroderma, causing digital amputation.

Pathology.—According to Lewin and Heller, the principal alterations in the structure of the skin affect the corium. These consist in dense fibrous bands running in all directions, having a peculiar homogeneous appearance, so that it is impossible to differentiate the individual fibrillæ. It is probable that the enlargement depends on the absorption of a coagulable albuminoid substance. The number of capillary vessels is notably lessened. As a sequence of the diminution there is distension of the venous system. This may explain the production of the violet circumferential areola. Unna has found the lymph channels and lymph spaces very much narrowed. It is generally admitted that lymphatic obstruction plays an important part in the production of the diseased condition. There is lymph as well as blood stasis, and this brings the disease into relation with the urticarial wheal, while the arrangement in course of a nerve track recalls zoster.

Symptoms.—It occurs in two forms, which, though in well-marked examples sufficiently distinct, present occasionally a mixed type.

DIFFUSE SYMMETRICAL SCLERODERMA.

Exposure to cold or damp, with pains in the joints, ascribed to rheumatism, may precede the actual onset, or this is of such an insidious nature that only a sensation of stiffness in some part of the body, the neck frequently, at least in the upper half, is the earliest symptom to attract attention. A feeling of tightness is experienced, increasing in degree and area, the skin is dry, harsh, wooden, but there is no very defined line of demarcation between the sclerosed and healthy regions. The deeper parts of the skin are invaded, the muscles may be implicated, the integument as a whole is over-distended, the infiltration into its substance causing the hardness and immobility. There is no pitting on pressure, motion and expression are alike abrogated in the affected areas, sensation is uninfluenced, and temperature is only slightly lowered. Occasionally in place of induration a hard œdema appears.

Consequent on this, the hypertrophic stage, there may be further changes. White ivory-like patches of varied size appear round these there may be a delicate violet or pinkish halo of dilated capillaries, individually invisible, and in addition a degree of pigmentation. Little telangiectatic dots or tufts may be studded here and there. The sebaceous secretion and the perspiration are in abeyance, hence the surface is apt to be scaly, and itching is complained of. The mucous membranes sometimes participate, and even deeper structures, as the tendinous sheaths or even the pleura, may apparently suffer. In the infiltrated form the tendency is in time to gradual recovery, though this may not be quite complete. In the œdematous and also in the infiltrated, though less often, there is much greater liability to shrinking and atrophy, from which recovery is seldom perfect. Death may result from fixation of the thoracic muscles, contraction of the diaphragm even being finally arrested, as in a case recorded by me.

CIRCUMSCRIBED SCLERODERMA OR MORPHŒA.

The lesions are more commonly non-symmetrical than in the diffuse form, and are generally more localised and defined. Sometimes a lardaceous spot or patch, or several, show themselves, surrounded by a violaceous or lilac areola. In other instances there is a purplish or reddish blush, within which after a time the skin assumes in part a yellow tint, with a waxy surface. Such spots or streaks often occur at intervals in the track of a cutaneous nerve, hence the term "herpetiform" applied by Hutchinson. The sensation imparted to the finger may be that "of a piece of parchment embedded in the integument." The redness in the early stage is eventually transformed into a dark brown pigmentation, which with the condensed tissue becomes in turn absorbed. The disease in this form has less tendency to progressive advance than in the diffuse.

Diagnosis.—The diagnosis should not present much difficulty in the diffuse form. It may be confused with diffuse or disseminated lenticular cancer of the skin, when, as occasionally though rarely happens, this is primary. But in it the colour is of a redder or reddish brown tint, the tendency to ulceration, which only occurs in scleroderma when over a joint, or at the point of a finger, is more marked; there is severe or stabbing pain, and the malignant cachexia manifests itself, in contrast to the comparatively good state of the health in scleroderma. Chronic localised eczema on the leg may appear like a patch of localised scleroderma, but the immediate improvement when proper treatment is instituted will clear up the difficulty. In leucoderma there is no induration.

Treatment.—Our efforts should be directed to promote the inherent tendency to resolution. Warm baths, massage, frictions with salicylic vaseline, kneading the parts, the avoidance of chilling the surface, and the administration of small doses of iodide of potassium, for a prolonged period, with intermissions, comprise the principal means in our power.

SCLEREMA AND ŒDEMA NEONATORUM.

These conditions, which have been often confounded, have nothing in common with scleroderma. The first recorded instance of sclerema was that of Usenbensius in 1718. In the end of the eighteenth century Underwood wrote about it, but confused it with erysipelas. Since both diseases are of great rarity in Britain, the chief sources of information have been Italian and French physicians, and among these Somma and Parrot may be specially mentioned. The most complete statement of all the phenomena is that of Ballantyne.

SCLEREMA.

This affection may be congenital, or develops within a few days of birth. If not actually presenting the characters of prematurity, the infant is either weakly at birth, or soon becomes so. The most constant and striking symptom is coldness of the surface, described as like marble. Rigidity is another, principally seen in the difficulty of moving the limbs.

The skin cannot be pinched up or caused to slide, and looks as if stretched, yet induration is not always demonstrable. Œdema and induration may be found in the same patient. The colour varies from yellowish to livid, bluish red, or even brick-red. It commonly begins in the feet, cheeks, or hands, and spreads from thence, but the integument of the front of the chest usually enjoys immunity. Both the pulse and respiration are slowed. There is a peculiar, weak, squeaking cry. The course is rapid, and it commonly ends fatally, but instances of recovery have occurred.

Œdema Neonatorum.

This is probably, as Ballantyne holds, not a disease *per se*, but rather a phenomenon met with in connection with other maladies, one of the symptoms in fact of cardiac, pulmonary, or renal disease, or of blood changes. There is no fall of body temperature, sometimes indeed a rise. The child exhibits great drowsiness, and dies of diarrhœa or convulsions.

The microscopic appearances in the indurative form consist principally of an increase in the connective tissue of the subcutaneous layer. A small cell infiltration of all parts of the integument has been found. This is very likely the primary, the increase in the connective tissue a secondary change.

Ballantyne thinks that "in most cases the morbid process resulting in sclerema is initiated in utero, that the most important factor in its pathogenesis is an antenatal lesion of the nervous system, possibly of the thermic centres, and that cutaneous chilling is only one of several conditions that may serve to develop the malady when it is not already present at birth."

Treatment.—Warmth is the essential element, and the best results are to be obtained from the use of an incubator. The next best mode is to envelop the infant in cotton-wool. If possible, the child should be nursed; if unable to swallow, it may be fed with a spoon, or œsophageal tube and glass syringe. Warm baths, oil inunction, massage, and passive movements of the limbs, are advantageous.

ELEPHANTIASIS.

A doughy, chronic, indolent enlargement of a limb or some other part of the body.

Etiology and pathology.—There is lymph stasis and increased growth of fibrous tissue. The source is to be sought, according to Unna, not so much in occluded lymphatics as in a resistance to the return of blood. This view is supported by the somewhat analogous condition found in phlegmasia alba dolens. "Elephantiasis develops from an œdema of the skin, which persists after the healing of erysipelas, and is to be ascribed to the persistence of gradually organising thrombi in the veins." Either the erysipelas is not completely recovered from, disturbances of circulation remain, absorption is deficient, probably all the streptococci do not perish, or fresh infection occurs.

Symptoms.—The disease occurs in two forms—one, that met with in tropical climates, largely of filarial origin, and dealt with in another portion of this work; the other occasionally seen here, and usually a sequence of repeated attacks of erysipelas. Perhaps the commonest variety is that affecting the loose skin under the lower eyelids. In marked cases, two symmetrical, cushion-like, soft prominences arise above the malar region, not painful unless inflamed. Or a similar condition may implicate the upper lip. Or one leg or foot, or both, undergoes such hypertrophy. The limb may become enormously enlarged, due to overgrowth of the integument and connective tissue. During periods of quiescence, or in an early stage, the colour may differ little from normal, but in time warty protuberant masses appear, fissures and ulcers break forth, intertrigo occurs between the folds, and a heavy, disagreeable odour is exhaled. The weight and stiffness so occasioned give rise to much distress. Eventually the mass of tissue while it increases becomes denser. The limb assumes a deep red hue with more or less pigmentation. Varicose lymphatics are seen on the surface.

Treatment.—In the treatment, massage, bandaging, and electricity may improve the state of the limb, at least temporarily. Gibson has recorded an instance of the solid subocular œdema cured by injections of anti-streptococcic serum, but this failed to benefit another. Prevention should be aimed at, by treating erysipelas by strong ichthyol ointments, a plan which gives much comfort to the patient, and at the same time in many cases obtains a rapid cure.

ANOMALIES OF PIGMENTATION.

The pigment found in the human skin has been divided into melanin or iron-free, and hæmosiderin or iron-containing. This serves to connect the latter in its origin as derived from the blood, while the former, as met with in the freckle, is more obscure in its source. Much is apparently formed in the corium, and is conveyed to the deeper layers of the epidermis either by the agency of mobile cells or through the lymph channels. The colouring matter of the skin may be in excess or deficient, and each may occur congenitally or be acquired.

Congenital excess takes the form of *pigmentary moles* or *nævi*; these may be simply dark brown stains, or raised, with smooth or uneven, warty, or papillary surface, or in addition bear strongish hairs. Their growth increases with that of the skin. They are chiefly met with on the face, neck, and back, and their importance consists in that, as life advances, they are apt, often as the result of irritation, to become the starting point of melanotic sarcoma or other varieties of malignant tumour.

Congenital deficiency constitutes *albinism*, in which there is total absence of pigment in the skin, hair, and eyes. This is liable to have associated with it some degree of mental and bodily feebleness, though to this there are exceptions. Partial cases are seen occasionally. Heredity in marked degree can usually be traced, with family prevalence.

The treatment of moles is either excision, if small or threatening to degenerate, or by electrolysis much as in the removal of hairs: nothing can be done for albinism.

Acquired excess may be idiopathic and limited, as the *freckle* or *lentigo*. These are commonest in red-haired persons, on the face, next in frequency on the arms and hands. They seldom appear before the age of 6, are pin-head or lentil sized, but may form larger aggregations, and are yellowish brown in tint. They assume a brighter hue in summer, do not wholly fade in winter, but often disappear in old age. *Chloasma* is the term used both for idiopathic more extensive pigmentations and for those of symptomatic origin. Thus such may follow counter-irritants, exposure to radiant heat, as on the shins of stokers, or even from extreme cold. Friction, pressure, and above all scratching, occasion it. The symptomatic variety may be the concomitant or sequel of eruptions, as those of syphilis or lichen planus, more rarely psoriasis. In pregnancy, besides the darkening of the mammary areolæ, patches of pigmentation sometimes appear on the forehead or elsewhere, to fade again after parturition, though probably recurring under similar circumstances. Cachectic discolorations are met with in cancerous tumours, in phthisis, and Addison's disease. Arsenic long continued may occasion in some brown discoloration, which slowly disappears if its use is abandoned. *Argyria,* a slaty blue tint resulting from the administration of salts of silver, is more permanent. The same hue has been seen in connection with melanotic sarcoma, apart from silver.

The removal of such stains is difficult. Freckles may fade when lightly touched by a needle attached to the negative pole in the electrolytic circuit. In ease of larger patches, perchloride of mercury, cautiously increased from ½ gr. to perhaps 5 in the ounce of almond emulsion, sopped on two or three times daily, may cause them to vanish; or peroxide of hydrogen used in the same way is sometimes successful.

Leucoderma or *vitiligo* is a form of misplacement of pigment not very infrequent. In females oftener than in males, and between the ages of 10 and 30, white, usually rounded spots appear, with darker margins as if the pigment had been chased outwards, to be heaped up at the edge, or the collateral bronzing may be uniform. There is no other alteration, but the process is apt to extend in area, and fresh patches to form. The neck is a common situation, but it may be anywhere.

Since the cause is unknown, rational treatment is impossible. The colour is seldom restored, though, as it is difficult to trace cases for many years, it eventually may be. On exposed parts temporary alleviation can be effected by staining the whitened areas with walnut juice.

XERODERMA PIGMENTOSUM.

A disease usually commencing in infancy, affecting the exposed parts, characterised by the successive occurrence of erythema, freckles, white shining spots with telangiectasis, warts, and carcinomata, with an eventual fatal issue.

Kaposi first described it in 1870; since then probably about seventy cases have been recorded, scattered over many countries. The disease shows a peculiar family prevalence, since usually only children of the same sex suffer. It begins as a rule in course of the first or second year, occasionally later. With or without the previous occurrence of erythema, freckle-like spots appear on the face. These grow more numerous, darker and more widely disposed, affecting in turn the neck, backs of the hands and forearms, rarely extending beyond the exposed parts. The skin becomes thin, dry, and rough, desquamating as fine flakes. There is ectropion, congestion, and watering of the conjunctiva. White shining areas appear scattered here and there, and in these dilated stellate or linear venous radicles. This partial sclerosis affects those parts which are most stretched, as the malar region, the nose, ears, etc. Flat, isolated warts show themselves as years progress, and if unchecked these assume the type of carcinomatous growths, which penetrate deeply, ulcerate, and wear the patient out.

The essence of the disease seems to consist in a congenital weakness of the skin to the blue and violet rays. The deposition of pigment, consisting of melanin granules, is probably an effort at protection. There is in places a hypertrophy and sclerosis of the connective tissue, with obliteration of the capillaries, and consequent collateral venous stagnation. The carcinomatous formation at so early an age may perhaps be due to the rapid changes occurring in the skin in this disease.

A somewhat similar condition has been described by Unna under the name of "sailor's skin," as it manifests itself chiefly in the weather-beaten. On the ears, cheeks, temples, and hands, a diffuse cyanotic redness appears, followed by mottling and reticular pigmentation. Between this there are non-pigmented areas, while the skin becomes rough and hard, even warty. Lastly, the sebaceous glands hypertrophy, and papillary and ulcerating carcinomata develop.

Treatment.—The treatment applicable is the sedulous shielding of the integument from sunlight, especially in summer. This may be effected by painting with lotions of a bland order containing raw umber, and by wearing brown veils and gloves. The warts should be destroyed by salicylic acid collodion, or excised. The conjunctiva should be bathed with boric acid lotion.

STRIÆ ET MACULÆ ATROPHICÆ.

Glistening, pearly white, or bluish coloured thinned lines or spots often occur. As a consequence of over-distension of the integument, from excessive obesity, or in the course of pregnancy, or from too rapid growth in weakened states of the system, the skin yields in the lines of normal cleavage, leaving distinct gaps in its substance. There is a primary stage of irritation when the tint is reddish, passing finally into one like mother-of-pearl. The condition may be very widespread, as in a case recorded by Taylor, where long and deep lines arose on the trunk and limbs of an obese mulatto girl of 17. Something similar may follow the absorption of patches of localised scleroderma. The skin becomes converted into an attenuated fibrous membrane, covered by a thin layer of epithelial cells.

NEW FORMATIONS.

MOLLUSCUM CONTAGIOSUM EPITHELIALE.

ROUNDED, mistletoe-berry-like papules or tubercles, whitish or pink, from a pinhead to a pea in size. Bateman apparently first described and figured it; Paterson of Leith proved its contagiousness: Hutchinson, Pick, Duckworth, and others have contributed to our knowledge.

These growths are never met with on the palms or soles, are most common on the face and eyelids, but occur also on the neck, trunk, limbs, genitals, and scalp. Rarely they have been seen on the red portion of the lips. In shape they present some resemblance to a miniature mother-of-pearl shirt button, a waxy or warty horny prominence, rising abruptly from the skin. The top is flat or slightly cupped, with one or more openings leading into the interior, from which, at least in the larger specimens, some whitish material can be expressed. Their consistence is firm and solid, and their colour either that of the skin or a shade of pink. Sometimes a reddish areola surrounds each. Though usually sessile, they may become pedunculated. Various changes may happen; they may inflame and ulcerate, the base taking on a degree of induration. From the inner surface thus extruded, a cutaneous horn may sprout. Often a moderate itchiness accompanies them. Not very common, it is chiefly seen in young persons. After a duration of uncertain length, many months perhaps, it spontaneously disappears. The disease is undoubtedly communicable. Pick, by inoculation, has shown that the earliest traces become apparent in about three months, and as many more elapse before the customary size is attained. Numerous cases have occurred in frequenters of the Turkish bath.

The nature of the ailment has been the subject of much discussion, its structure has a resemblance to that of an acinous gland, but, as Unna remarks, we do not find remains of sebaceous glands in the little tumours, nor evidence of transitional stages. Though it is only found where such or analogous labial glands occur, it would seem that it arises in the prickle layer of the epidermis. There is a down growth and extension of separate epithelial areas, and these, by space exigencies, are forced into the gland-like shape. In the central portion part of the prickle cells are converted into a colloid mass, part retaining their reticular texture; these are the peculiar bodies regarded by some as animal parasites, psorosperms or coccidia. The medium of contagion has not yet been isolated.

Since the disease will spontaneously disappear, even though only after a time, it is probably the best treatment to touch each once or oftener with pure liquid carbolic acid, which causes them to wither without leaving any trace.

DARIER'S DISEASE.

A disease which seems to bear a degree of relationship to molluscum contagiosum, referred to by Wilson as ichthyosis sebacea cornea, but of which Darier, in conjunction with Thibault, gave a clear pathological account. Only a dozen cases have been recorded. It begins on the face or trunk, but extends from thence, reaching its acme in the inguinal folds and axilla or scalp. The earliest lesions are pinhead-sized firm papules, pale at first, but becoming purplish or brownish red. These flatten, and become covered with a plug-like crust, firmly adherent, and brownish, grey, or yellowish. This on removal is seen to be embedded in a funnel-shaped depression, bounded by an annular border, generally corresponding to a pilo-sebaceous follicle. The palms are dry, the skin tense, and the nails distorted. The elements multiply, scratching may cause bleeding, horny projections may arise; or, in parts such as the groin, vegetations appear, exuding an offensive pus, or covered with malodorous accretions. The health is little affected, and the condition is a chronic one. There are affections which look like mild examples, such as have been described by Colcott Fox as *acne scrofulosorum*, or by myself as *impetigo varioliformis*. These usually occur in children, the eruption consisting of flat, small papules, with a central sulphur-yellow, conical, firm crust. The disease is to some extent contagious. Bodies, in aspect like coccidia, are found in the epithelial cells, and though opinion as to these is still divided, the preponderance is in favour of this being simply a form of hyaline degeneration, analogous to that in molluscum contagiosum.

The treatment of the cases reported has not been successful. The indications to be followed are the employment of prolonged warm sulphur baths, and the use of resorcin in soap or as a pretty strong ointment.

XANTHOMA.

Spots or nodules of the skin, assuming various shades of yellow, may be localised particularly on the eyelids, or generally distributed, and then sometimes found on the mucous and serous membranes, which develop slowly. It was first described briefly by Rayer, more fully by Addison and Gull. Hutchinson has contributed largely to our knowledge. There is a variety or related disease met with in association with glycosuria, only definitely recognised within the last fifteen years or so.

Etiology.—Xanthoma of the eyelids is more common in females; it exhibits decided family prevalence, and may be hereditary. Hutchinson regards it as a direct consequence of frequent disturbance of pigmentation of the eyelids, by whatever cause produced. Repeated attacks of migraine have in many instances preceded it. In generalised xanthoma non-obstructive jaundice has been present at some time, in a proportion of cases, in others hepatic enlargement, digestive troubles, and lithiasis have been noticed. The relation to diabetes is undeniable; it may be, as Török and Johnston hold, an irritative process, the irritation being supplied by the excess of glucose, or some faulty product of metabolism circulating in the blood.

Pathology.—Pathologically, in xanthoma of the eyelids we find, according to Unna, the cutis transformed into perivascular cords, which, separated by large lymph spaces, lie loosely over one another. Within the lymph spaces there is a special form of fat, consisting of very small elements. These, when closely packed, give rise to mulberry or fish-roe-like conglomerations, the so-called xanthomatous masses. There occur also numerous spindle cells, with large, single, vesicular nuclei. In xanthoma multiplex and diabeticorum there are large cellular areas, with no special relation to the vessels, which undergo a central fatty change. In the diabetic form the inflammatory changes are more marked.

Xanthoma Palpebrarum.

This, the most frequent form, is characterised by the appearance of yellow patches on the eyelids. These manifest themselves usually in adult or middle life, but may occur in childhood. Their earliest situation is near the inner canthus of the left eye, gradually increasing and extending till a semicircle is produced. The right eyelid is secondarily invaded, a fairly symmetrical arrangement being eventually assumed. The individual lesions are flat or slightly, exceptionally markedly, prominent, soft, smooth, and well-defined. The lids are commonly pigmented. They give rise to no subjective sensations. Having reached a certain size, they cease to enlarge, but do not disappear.

XANTHOMA MULTIPLEX.

This is a rare form, and is encountered both in children and adults. It may commence on the eyelids, extend to them, or leave them intact. The yellow spots and areas are scattered over the body, in particular on the flexures of the palms, soles, elbows, knees, and joints of the fingers. On these latter situations a tuberculous in place of a flat patch, with fine vascular tufts on it, may develop. The skin has frequently a generalised yellow tint. Tubcrous or giant forms have occurred. It is possible for the disease to disappear completely.

XANTHOMA DIABETICORUM.

Though most of the cases, amounting to rather over twenty, which have been recorded, occurred in persons whose urine contained more or less sugar, this was not invariable. The patients were, however, usually obese. The eruption first shows itself on the outer aspect of the extremities, especially of the forearm. It appears suddenly, spreading continuously or intermittingly to other regions. It is found on the knees and elbows, nates, and mucous membrane of the mouth, not on the eyelids apparently, unless a peculiar case reported by Darier is accepted as purely this form. While no special sensations precede its appearance, when developed, marked pruritus or tenderness is complained of. The lesions are firm, discrete nodules, wholly reddish or pinkish, or with a yellow point in the centre. They are seated at hair follicles as a rule. After an indefinite period the eruption vanishes, leaving no trace, but it has been known to relapse concurrently with an aggravation of the glycosuria.

Treatment.—Owing to the situation, excision is inadmissible in xanthoma of the eyelids; improvement has been reported by electrolysis. Partial success has, according to Darier, followed the application of perchloride of mercury in collodion. Besnier recommends the long-continued administration of turpentine internally. In xanthoma diabeticorum, all remedies which act beneficially on the glycosuria, favour the disappearance of the eruption. Codeine, in doses to suit the case, and menthol soap, with an ichthyol lotion, soon caused the irritable spots to wane in a case of mine.

COLLOID DEGENERATION.

Wagner first described this rare condition as colloid milium in 1866. Since then cases have been reported by Besnier, Liveing, Feulard, and others. This apparently may occur at any age, and gives rise to no subjective sensations. It consists of nodules, usually localised on the upper half of the face, but occasionally developing on the neck, upper part of the chest, and arms. They may be from a pinhead to a pea in size, are hard to the feel, embedded in the cutis, of the same colour as the skin, or yellowish or brownish red. On the face, at least, they have a translucent appearance, which causes them to look like vesicles. If incised or squeezed, a transparent gelatinous mass is extruded. Phillipson has shown that it is an epithelial overgrowth which has undergone a colloid degeneration, but Unna thinks the process a more complex one. In treatment, if desired, electrolysis might be tried.

TUBERCULOUS AFFECTIONS.

The discovery of the bacillus of tubercle by Koch, and its subsequent identification in quite a class of cutaneous diseases, has modified our conceptions with regard to a number of skin affections. The best known and the commonest is lupus vulgaris.

Etiology.—All forms of tuberculosis of the skin are, as regards their commencement, most prone to attack the young; lupus, at least, originates principally in childhood. It is more common in females than in males, and from the fact that its earliest situation is generally an exposed part, the germs may have been implanted in a minute lesion of continuity. There are occasional instances where the disease is widely spread, or nearly quite symmetrical, the individual patches being coeval. These are difficult to explain on the inoculation theory. The general health in lupus patients is often apparently excellent, yet such are seldom long lived.

Pathology.—Unna has shown that the lupus new growth may be placed under one of two categories, that circumscribed and tending to encapsulation; and that which is diffuse and tending to extension. While bacilli, indistinguishable by any of our present methods from those of tubercle, are found in sparing numbers in the giant cells of the lupus nodule, it is probable that these are not the only forms under which the infectious agent presents itself; Hallopeau states that zooglœa have likewise been discovered. It is widely accepted that in these organisms we have the cause of irritation. The nodule is composed of a delicate reticulum containing a mass of cells with granular protoplasm and large vesicular nuclei. These are deeply set in the cutis, of which the vessels and fibrillar structure are by them pushed aside. Giant cells arise inside this mass, which, as it increases by cell proliferation, exerts pressure on the surrounding healthy corium, thus favouring encapsulation. Within the giant cells the bacilli may remain imprisoned and dormant, but not dead, for an indefinite time, thus accounting for recurrences or fresh outbreaks after the lapse of years. Some skins, however, offer a greatly diminished resistance, and the cells of the lupus granuloma infiltrate themselves along the vessels, and insinuate themselves into the lymph spaces. This is typically seen in the florid and erythematous forms, in which, clinically, it is difficult to discover nodules. The constituents of the bacilli alone or their toxines may occasion a high degree of sero-fibrinous inflammatory action in the neighbourhood of the lupus tissue, giving rise to exudation and crust formation. This occurs chiefly in the diffuse form. It is a disputed question, whether, in ulcerated lupus, there is a mixed infection with pyogenic organisms in addition to the bacilli, or whether the tuberculous poison singly possesses the liquefactive power, which, in excess, gives rise to rapid destruction, comparable to the phagedæna in syphilis. In some instances, a hardening of the lupus infected area arises, proceeding from an increased growth of the normal adjacent connective tissue, and this, while it tends to obliterate the lupus new growth, leads also to a keloidal condition in the imperfect scar, shown as bands or ridges, seen frequently after scraping operations. In tuberculosis of the skin, the bacilli are much more abundantly present than in lupus, and this may partly explain the greater destruction within a given area; this is, however, partly due to the weak state of the tissues in such cases. The tuberculous gumma has its source in a tuberculosis of the lymphatics of the subcutaneous tissue.

Symptoms.—The clinical features must be considered under the different varieties which occur.

LUPUS VULGARIS.

The primary eruption in lupus, the determination of which at any stage enables one almost with certainty to recognise it, consists of nodules, the size of a pin-head or millet seed, of a yellowish brown, somewhat translucent aspect, deeply embedded in the corium, and characterised by remarkable indolence and persistence. Slightly prominent, their surface is smooth. The nodule may be masked by hyperæmia, but if this is dispersed by pressing with a glass plate, Unna's diascope, it comes into view, though somewhat blurred at its margins. Though occasioning no subjective sensations, there is a degree of tenderness elicited, when the part is squeezed. The softness of the growth is shown by the readiness with which a probe can be made to penetrate it.

These nodules, by their increase in number and aggregation, produce patches of irregular outline, and raised above the surface. If extending, a zone of congestion surrounds it. The whole area is not composed of lupus tissue; part is a species of sero-fibrinous inflammation, with a greater or less amount of concurrent vascularity, a feature strongly marked in certain cases. If the patch be of long standing, it may include islets of scar, and its colour may be a dull purplish tint from venous stagnation. The general appearance of the surface is glossy, but flakes of cuticle adhere to it, and tend to scale off. In other examples part of the patch bears crusts, which, though chiefly attached by their margins, are rather hard to remove. Beneath them there is either a raw, exuding, granular bleeding space, or an excavation filled with purulent fluid.

There may be but one or many such patches, arranged irregularly on any region. The face and extremities constitute the commonest situations, the scalp the rarest. The tendency is towards slow though sure extension, and this too often in spite of well-devised methods of treatment. One or more patches may unaccountably wholly become absorbed, and leave merely a smooth white cicatrix, while others go on growing larger. In the end a pink inelastic tissue takes the place of the lupus, apt in itself to become afresh the seat of lupus, hence not a sound scar. This may in time acquire a degree of whiteness and pliancy. It is not so prone to shrink with age as scar tissue in general.

Lupus may assume the *serpiginous* type, in which the nodules are arranged as a band from a quarter to half an inch broad, exposing a crenated and advancing edge, which presents a well-defined erythematous halo. There is usually a good deal of crusting on the surface, but in its wake is scar tissue. It is more frequent on the neck, trunk, and extremities than on the face.

Another variety is *lupus verrucosus.* This is principally met with on the knuckles, back of the hands, wrists, and feet, but occasionally elsewhere. There is added to the lupus new growth, epithelial hypertrophy, associated with elongation of the papillæ. There is warty excrescence superadded to the lupus patch, which here takes the form of a dusky red or violet tubercle. Some of these remain smooth, others in part become warty. Pus can often be made to ooze from the tubercles, or from between the papillomata. There is not much pain, more a feeling of tension from the limitation of movement. It extends slowly at the periphery, but also by formation of new foci, and heals with the production of a white scar. Closely allied to this is the post-mortem wart, *verruca necrogenica*, caused by handling animal tissues infected with tubercle. It is thus found in butchers and cooks, as well as in those conducting frequent autopsies. There is first a small red, smooth nodule, on which a pustule, and subsequently a warty papilloma develops. It may persist indefinitely.

Various complications may occur. Thus, especially on the upper lip, solid lymphatic œdema, giving rise to *lupus hypertrophicus*, is seen. Or the cellular lupus growth is trivial in comparison with the widespread persistent erythema associated with it. Though, in general, the characteristic nodules can be made visible by stretching the skin, yet, as it sometimes assumes the butterfly shape on the nose and cheeks, it has been termed by Leloir, *lupus vulgaris erythematodes*. Again, soft, papillary, readily bleeding, spongy vegetations may sprout from the base of the lupus ulcer, as from those due to other causes. The bones and periosteum may become diseased, or conversely, lupus of the skin may be secondary to tuberculous disease of bone. Erysipelas and lymphangitis may attack at any time, and while a temporary improvement follows, this is succeeded by increased rapidity of spread, and also by a condition of elephantiasis.

MILIARY TUBERCULOSIS.

This is seldom encountered save in those suffering from advanced tuberculosis of internal organs. There is an absence of the yellowish brown nodules of lupus, while it is found close to those orifices of the body where skin and mucous membrane join. As usually seen, there are shallow ulcers, exuding a scanty secretion, and with a reddish yellow granular floor.

Under the term *lichen scrofulosorum* Hebra described an eruption which, if not yet definitely proved tuberculous by the discovery of the bacillus, is at least one occurring in the scrofulous, or persons predisposed to this class of ailment. It is characterised by brownish red papules, about the size of a pin-head, chiefly localised at the mouths of follicles, covered with fine scales, or bearing a minute pustule, though some are shiny. They pursue a chronic course, are scattered in groups or circles on the back and abdomen, and, after persisting for months, disappear. Only moderate itchiness accompanies them, but the lymphatic glands in the neighbourhood are usually enlarged. Seborrhœic eczema of the scrotum or groins may be associated with it, or acne-like pustules may form on the legs. It is seldom encountered save in childhood or adolescence. Sack and Jacobi regard it as a mild manifestation of tuberculosis, Besnier as a form of folliculitis, special to the scrofulo-tuberculous, but it is not entirely follicular in situation. The treatment originally suggested by Hebra is always effectual. This consists in the application of cod-liver oil externally and its administration internally.

THE TUBERCULOUS GUMMA.

This is most common in the skin over inflamed glands in scrofulous persons, but it may occur independently as subcutaneous nodules, which enlarge, encroach on the integument, liquefy and burst. The resulting ulcer has the characters of the strumous sore. The edges are thin and undermined, the base covered with flabby, indolent granulations. There is little tendency to heal, but if it does, a puckered scar is produced.

Diagnosis and prognosis.—In the diagnosis of lupus it is most important to search carefully for the primary, yellowish brown, painless nodules, either by stretching the skin, or by examining it through a plate of glass, which blanches it and brings them into relief. Its slow progress and usual history of a commencement in early life come in as auxiliaries. From tertiary syphilis the discrimination is not always easy. This in the inherited form may commence early, but the rate of advance is rapid when compared with lupus, and the test of treatment is available. Rodent ulcer rarely begins before 30, is seldom multiple, and originates as a semi-translucent nodule, or as a wart, or sebaceous accumulation, on the upper half of the face. Lupus itself in exceptional cases may undergo a cancerous change. The prognosis depends mainly on early recognition, and the institution of active treatment, when the disease is circumscribed. Unless this is done, extension is certain.

Treatment.—If the patch of lupus is small, it may be got rid of by careful excision, or, after thorough scraping, the surface may be cauterised with fuming nitric acid. Should catarrhal complications, shown by breaking down and crusting, be present, scraping again gets rid at once of a mass of unhealthy tissue. This procedure, however, is inapplicable, or at least is too radical a measure when the nose is affected, a situation where it is most desirable to save any remnants of epithelium as foci for cicatrisation. Here, multiple scarification, a process of cross-hatching, with a fine knife, repeated time after time, gives better final results. In all these instances healing is best promoted by dressing with pieces of Unna's zinc ichthyol salve muslin. The parts may now, or from the first, if the surface is unbroken, and particularly if extensive, be treated with various strengths of Unna's salicylic and creasote plaster muslin. Pieces are to be closely applied daily, commencing with the higher percentages, and tapering off with the lower, the surface being carefully cleansed at each renewal, and the treatment being persevered in till the white and soddened epidermis seems studded with a series of small red pits, or the pain complained of, which is at first intermittent, becomes too severe to be borne. The zinc ichthyol salve muslin is substituted till healing is complete, when the cycle may be repeated. This may be alternated with other methods. If the patches are numerous and extensive, painting them with pure carbolic acid, at intervals of a week or ten days, may be resorted to, a few only being treated at a time, to obviate danger of absorption. Or, as has been recently suggested by Unna, they may be painted, at intervals of a few days, with the liq. antimonii chloridi. If few in number and small in area, a 10 per cent. pyrogallic acid ointment in vaseline, applied twice a day for four or six days, may be used, then the salve muslin or zinc ichthyol ointment, till healed. Exposure to the X-rays at intervals, with due precautions, has recently been found to produce improvement, but whether a definite cure can in this way be obtained, it is as yet too early to determine. Much the same may be said of Finsen's light treatment, as carried out at his Institute at Copenhagen. Both methods appear to act more directly on the soil than on the bacillus. By these means the number of the nodules is gradually reduced, till in favourable cases they can be distinctly isolated. Each may then be bored out with a dental burr, and a pointed piece of wood, dipped in acid nitrate of mercury, inserted, or the point of a fine Paquelin cautery. For lupus verrucosus the salicylic collodion, or a pretty strong specimen of Unna's salicylic and creasote plaster muslin, applied and changed twice daily, best removes the warty accumulations. The case is then reduced to the condition of simple lupus, and can be dealt with as such. From the state of the patient, cure of miliary tuberculosis is scarcely to be expected. The tuberculous gumma, when fluctuation is perceptible, should be opened and scraped out. The successful treatment of tuberculous affections of the skin needs great patience and perseverance on the part of sufferer and doctor.

LUPUS ERYTHEMATOSUS.

A disease in nature allied to inflammation, since there is cellular infiltration round the vessels, but characterised clinically by the formation of erythematous discs or patches, usually with a raised margin and scaly, but sometimes smooth, depressed centre, leaving more or less permanent scarring. Though Hebra seems to have been the first to offer a description under the name of seborrhœa congestiva, believing that the morbid process commenced in or about the sebaceous glands, Cazenave more fully recognised the disease, and formulated an account which, in the main, holds good even yet. He is responsible for the appellation lupus erythematosus, which, though possibly technically incorrect, is nevertheless so long established that neither erythema atrophicans, suggested by Morris, nor ulerythema proposed by Unna, nor several others, have been able to displace it.

Etiology.—It has been met with in children, is most common in early adult life, but may appear in middle age. It may fade and relapse, or, after persisting for an indefinite period, it disappears, its previous situation being evidenced by a white and thinned condition of the skin, an appearance, however, frequently seen while the disease is in an active state, in certain of the patches. It prefers parts specially liable to sudden variations in blood supply from their distance from the heart and outlying situation, with often a feebleness in the peripheral circulation. Women are more frequently attacked than men; its subjects not rarely die of tuberculosis or belong to phthisical families, while sometimes tuberculous glands are found in the neighbourhood of the patches. At the same time the connection between tuberculosis and lupus erythematosus is, for the present, more a matter of inference than proof.

Pathology.—Pathologically, it is an inflammation of the skin, with marked infiltration of mononuclear cells round the dilated blood vessels. There is œdema of the tissues, with distension of the lymphatics. The elastic tissue is preserved. There is primary hyperkeratosis, sometimes epithelial growth. Peculiar scales are formed, with horny pegs, which plug the follicles. Finally, there is atrophy as a result of the long standing morbid changes. The disease does not, as was believed by Hebra, necessarily begin in the glands.

Symptoms.—The eruption at its commencement is not unlike psoriasis. There are small yellowish brown or purple-red spots, with a well-defined edge, and a slightly depressed centre, which, though smooth at first, soon becomes invested with dry, greyish, splintery scales. These are firmly adherent, but if forcibly removed, have thready tags beneath, drawn out of the hair follicles. Further advance may be due to the appearance of new spots, or to the increase in size of the original ones, or to both. Horny comedones may be seen near the margins. The large part which erythema plays is manifested by the almost total fading under pressure. Symmetry of distribution is the rule. The nose and adjacent portions of the cheeks are often so affected as to convey the idea of a butterfly; the lobes of the ears—a very characteristic situation—the hands, and rarely the feet, are the localities most frequently attacked. Complaint of itchiness or of burning sensations may be made, frequently merely of disfigurement.

So superficial may the process be, that it seems only a persistent erythema; or a considerable amount of plastic thickening exists. The latter approximates in aspect to lupus vulgaris. A rare variety is that named the *telangiectic* by Crocker. In it, interspersed among the more ordinary lesions, are areas where the capillaries exhibit considerable dilatation. Another, still more infrequent, is the *exanthematic*. Here, in addition to the ordinary sites, the trunk and extremities are invaded. There are large blotches of a deep crimson red, more or less completely covered with scales. A cachectic condition is associated with such, and they tend to end fatally. Unlike lupus vulgaris, it is often encountered on the scalp, leading to baldness. While common enough on the backs of the hands, it is sometimes seen on the palms, there it is very purely erythematous, and does not leave scars.

Diagnosis and prognosis.—Careful search for cicatrices is essential, as, if such are discovered, this would exclude eczema, psoriasis, and tinea circinata. This is particularly the case with the exanthematic form. Lupus vulgaris can generally be differentiated by the absence of the soft yellowish brown nodules. These are masked, however, in lupus verrucosus by the warty accretions, and on the hands there may be difficulty. When these have been got rid of, the nodules become visible. From chilblains on the hands, the persistence of lupus erythematosus during the summer, and the scaliness, will discriminate. The telangiectic cases look a little like acne rosacea, but there are no pustules and there is marked scarring. Crocker has described a case where it closely resembled lichen planus, but, while confusion as to the eruption on the hands is possible, lichen planus rarely, if ever, attacks the face. From a persistent erythema elevatum it may be nearly impossible to separate it. A recurrent erythematous syphiloderm is of more irregular distribution, and of shorter duration, when the area is taken into consideration. If on the scalp, the hairs must be examined to eliminate favus. Lupus erythematosus is notoriously resistant to treatment, therefore a guarded prognosis must be given; and though the disease, except in the exanthematic form, is not a dangerous one, still many observers have remarked that the subjects of it are not long lived, and are apt to be carried off by some intercurrent ailment.

Treatment.—Mild measures steadily persisted with are incomparably superior in their results to the employment of the active remedies so suitable in lupus vulgaris. The more purely erythematous, too, the individual case, the more necessary to avoid undue stimulation. Since there is at least an analogy with seborrhœa, if not association, sulphur in some form is often advantageous. Bulkley's formula, consisting of 1 drm. each of potassa sulphurata, sulphate of zinc, and glycerin, in 4 oz. of water, painted on three times a day, may do good. Or 1 drm. of glycerin to 1 oz. of the dilute liquor plumbi subacetatis, as suggested by Payne, similarly used, is applicable to many instances. Boeck has found thiol useful as—℞ Thiol, aquæ, mucilag. acaciæ, āā 1·0; ac. pyrogallic., 0·5—*M.* Linear scarification of the margins of the patches, repeated at intervals of about ten days, has a distinctly curative effect, and this is increased if, as W. Anderson suggests, the surface is painted subsequently with a 5 per cent. solution of carbolic acid. Internally, ichthyol, 3 to 5 minims or more, freely diluted, taken twice a day, does good. Quinine in full doses, as recommended by Payne, 5 grs. given from three to six times a day, according to the degree of cinchonism produced, persevered in for a long time, has proved valuable in rebellious cases. It must always be borne in mind that a spontaneous, if slow recovery is probable, and therefore caustics should only be used by skilled hands.

SYPHILIS.

A contagious malady, which, when its virus has been implanted, exhibits features allying it with the exanthemata, eruptions on the skin after a period of incubation, followed in some cases by late sequelæ, with immunity, in the large number of instances, against a second infection, and transmissible to the fœtus.

Syphilis probably dates from remote antiquity; but it appears to have, in historical times, spread on occasions widely, causing extensive ravages. Its intensity was at one time much aggravated by the excessive and injudicious employment of mercury. This led to a reaction against that drug, and its condemnation by some. The disease now shows much milder features on the whole, in part attributable to more rational management.

Etiology.—The characters are such as might with good reason be ascribed to a germ, but the proof that the bacillus discovered by Lustgarten is the causal agent is not complete. Introduction may take place at any part, though, for obvious reasons, this is most frequent on the genitals; sometimes the point of entrance cannot be discovered.

Morbid anatomy.—The morbid anatomy of the syphilides is comparatively simple. The changes mainly consist in an infiltration of cells in the neighbourhood of the vessels, which are themselves dilated, but the larger ones also present some degree of the alteration which has been termed endarteritis obliterans, a narrowing of the lumen in places owing to a fibrous increase in the adventitia or the media. The perivascular cell accumulation is lasting, so that, as Neumann and Unna have shown, histologically recognisable remains persist round the vessels after every syphilitic eruption. The induration of the chancre is due to a thickening and hardening of the tissue meshwork. The colour of the syphilides is produced, according to Unna, by a diminution in the connective tissue element, and by the perivascular infiltration. The blood shines through from the deeper parts; the amount of cellular investment of the vessels, combined with more or less stasis, determines the tint. Pigmentation is caused by changes in exuded hæmoglobin. Probably much of the eruption is occasioned by the action of toxines created by the as yet hypothetical germ. The occurrence of tertiary lesions is promoted by alcoholic indulgence, or by lowering influences, as privation or malaria; their locality is often determined by some injury. The view expressed by Unna that they always take origin in remnants of the primary or secondary tissue changes, cannot be unreservedly accepted, in view of the rarity of recurrence in the site of the chancre, and of their development in women who have received infection through the fœtus, but have never shown any primary or secondary symptoms. They are certainly most frequently seen in those who have been inefficiently treated by mercury during the secondary stage.

Symptoms.—About three weeks after infection, a nodule appears, indolent in nature, unless complicated by the presence of other animal poisons. It attains the size of a pea, but may be larger, and in course of ten days is a distinctly indurated disc, with a dark red centre, not liable to ulcerate. There are, however, many varieties of the chancre; the hardness is not invariable, may be indistinguishable on the female genitals, for example, or a colliquative, extending, destructive process may attack it—sloughing phagedæna. Coincidently with the development of local induration, there occurs, usually, enlargement and hardening of the neighbouring lymphatic glands—multiple adenopathy, as it has been called. This may not have become absorbed in a year, while the induration of the chancre may vanish in a few weeks, or last for several months.

Between the development of what have been termed secondary symptoms and the full induration of the chancre, an interval of two to four weeks elapses. Thus these do not manifest themselves till from seven to twelve weeks after infection. Slight malaise, feverishness, and evening headache, with nocturnal rise of temperature, are experienced. A degree of anæmia is observed, with languor and aching pains. If the skin of the abdomen and chest is about this period carefully examined in a clear light, in a cool atmosphere, a delicate, dusky, or pinkish mottling can be noted. This is very evanescent, is sometimes the sole eruption, and may pass off unperceived. Just about the same time there may be seen some congestion of the pillars of the fauces, symmetrical superficial ulceration of the tonsils, or even little milky-white patches on the inside of the cheeks.

So far the symptoms are tolerably uniform and simple, but from this period onwards irregularity of the manifestations is the rule, and one can never foretell in what fashion the subsequent phenomena may declare themselves. Certain general characters are, however, common to syphilides, and are of value in distinguishing them from the cutaneous eruptions which they simulate, for the imitation is rarely indeed perfect; to a careful observer it will be found lacking in some particular. Nor does the eruption, as a whole, preserve a constant type; the elements are incongruous; some degree of polymorphism is present in one region or another. It is an element of popular belief that specific eruptions are coppery in tint, or resemble raw ham, but this undergoes modification in blondes on the one hand, and in brunettes on the other, while in the old, or later in the disease, a brownish purple more nearly represents the shade. In certain localities, or should the skin lesions persist long, pigmentary staining remains after the changes which led to it are gone. While disposed bilaterally, usually symmetrically, they are often widely distributed, and have a tendency to occur in circles, or in irregular groups. There is seldom complaint of itchiness, or any subjective sensations, unless in cases where the eruption develops rapidly, and particularly when it takes the papular form. The rule of examining the entire surface of the body should never be forgotten if a syphilide is suspected; thus it may be possible to trace the gradual evolution of one form of secondary lesion from another, since such appear frequently in successive crops, overlapping each other in time. Inquiry into the history ought always to *follow, not precede*, the topical examination.

One of the commonest of the early secondary eruptions is that known as the *papulo-squamous*. It is sometimes mistaken for psoriasis, especially should the patient have reddish hair; more often, however, the error is the reverse, the simple ailment being held to be the specific. It is scattered as small spots from a pea to a sixpence in size over the body. These are usually covered with dirty whitish scales, very seldom showing any of the silvery lustre of psoriasis. The colour varies, but is apt to resemble that of ham, becoming darker the longer it lasts. Interspersed may be seen smooth nodules, or on the face, blotches as well. During retrogression the scaliness may be more pronounced, and the areas affected being larger, it appears more like psoriasis. When this in turn has been absorbed, and the patches have flattened down, a brown or grey stain is left, or there is a diminution of normal pigment, and a white macule is perceptible. Not so commonly in Britain, as on the continent of Europe, this form affects the palms as a symmetrical eruption. The thickness of the horny layer gives rise to more flattening, and a brownish red colour. The elements may be isolated, crescentic, or irregularly aggregated. Desquamation occurs in scales or flakes, and the eruption may still be traceable here when it has entirely faded elsewhere. While this or other of the earlier cutaneous manifestations are in progress, there is often considerable seborrhœa of the scalp, with patchy thinning of the hair, which at the same time is dull and lifeless, or there may be iritis. This eruption avoids the elbows and knees, favourites of psoriasis.

In situations where two surfaces are constantly or frequently in contact, or where glandular secretions readily accumulate, the moist papule or *condyloma* is found. If recent, these exhibit a more or less lively red colour, with a soft, velvety feel, not unlike plush. A thin, acrid sero-pus is shed over and around them. After a time they assume a dusky bluish tint, and acquire a drier surface. Considerable areas may be so invested, and auto-inoculation of opposing points occurs.

The epidermic hypertrophy may be accompanied by papillary overgrowth, so that a warty aspect, as if powdered over, is given to the papules; this has been called the *vegetating papule;* or the coating may be partly exudative, partly sebaceous, producing the *crusted papule.* Between, painful fissures may arise.

The *small papular syphilide,* if rare, is sometimes rather difficult of diagnosis. It varies from a pinhead to a linseed in size, is bright red at first, changing into a brown or dusky purple, with a shining top. They are numerous over the breast, abdomen, arms, and shoulders, more scattered on the back and lower limbs. A corymbose arrangement may be adopted, in which a series of small papules are pretty regularly set round a large. They disappear slowly, leaving pigmentation for a long time. Lichen planus is not infrequently mistaken for this. Careful search should therefore be made for any persisting traces of constitutional infection, though such may not be very evident, as the small papular syphilide is rather a late secondary lesion. Pustules may be found here and there, never in lichen. Itching and pigmentation are common to both, but the arrangement in the patch form is unusual in the syphilide. The papular, as indeed other syphilides, may be masked by the coexistence of some other skin disease, such as scabies.

The papular syphilide may be considered the basis out of which the *pustular* develops. Consequently there is a small and large variety. The greater the extent to which the papule undergoes this purulent liquefaction, the more severe the type of eruption. In some cases there is a *vesicular* stage which terminates in the pustular transformation. There may be merely an intermixture of pustules and papules, or the former may so preponderate as to constitute the most striking feature. The pus dries into a crust, beneath which is a superficial ulcer, healing with more or less scar. An inflammatory areola surrounds the pustule when at its height, replaced eventually by pigmentation. The eruption may be scanty or abundant, and dispersed over the body. It has been confused both with varicella and variola, but if there should be difficulty at first, the lapse of a little time will clear matters up. That variety of acne called varioliformis, limited to the face, especially the forehead and neck, presents some resemblance, but the restriction to these regions and the absence of comedones and of constitutional symptoms, are distinctive.

Rupia, though a form of pustular syphilide, generally occurs alone. The infiltration is more extensive than deep, the epidermis being soon raised as a purulent bulla. This by absorption becomes a crust, but the formation of pus beneath does not cease. The inflammatory area around extends, and secretion goes on from the enlarged surface. Thus the resulting scabs are progressively raised higher and higher, shrivelling up at the same time. A pyramidal pile of a dirty greenish black colour is built up, surrounded by a red and scaly ring. If removed, a shallow ulcer covered with a thin layer of pus is exposed. With fading of the areola cicatrisation, beginning in the centre, proceeds, though slowly. A white scar, ringed for a period with a brownish stain, permanently marks the site. This has been regarded as a late eruption, but it may appear from seven months to a year after infection, its early manifestation being evidence of a severe form of disease. It may be confounded with ecthyma, in which the pustules are indeed large and angry, but conceal an abraded surface, and leave a macule, not a scar. Rupia may pass into a chronic phase, spreading serpiginously and extensively.

In the form termed the *pigmentary syphilide,* yellowish or pale-brown stains, occurring independently of any other lesion which might have left them in subsiding, are seen, particularly on the sides of the neck in women, far less often in men. This is met with in the second six months of the disease, and vanishes very leisurely, hardly at all influenced by treatment.

The duration of time within which the symmetrical or secondary eruptions may appear is somewhat indefinite; all may have faded in six months or less from the infection, or traces, if no more, may be visible, even at the end of two years. In many cases, after the patient regards himself as cured, relapses or "reminders" occur. These, while preserving more or less the symmetrical character, are usually somewhat limited in distribution. They take the form of scaly patches on the palms or scrotum, or sores on the tongue. Should a generalised eruption manifest itself, it assumes the type of a scanty papular rash, or of a macular or roseolar efflorescence, in which the elements are few, of a paler rose tint, often imperfectly rounded or annular. They are fugacious, or yield readily to treatment, have no prognostic significance, though, as Fournier remarks, they exert a depressing and discouraging effect on the individual.

It is customary to divide syphilis into three periods—the primary, or that of local inoculation; the secondary, or period of generalised, symmetrical eruptions, which, be it treated or not, commonly terminates in not much more than two years at most. It may sometimes be prolonged by some of those irregular manifestations just alluded to. In many cases of syphilitic infection with this the disease finally ceases. But there is a residue, in which sequelæ, the so-called tertiary symptoms, declare themselves, after an interval of entire immunity, lasting from five to perhaps forty years. This constitutes the third period, and the lesions which then crop up exhibit no tendency to spontaneous cure, but, unless actively treated, steadily progress. They are mostly what are termed gummata, and invade the integument serpiginously. It must not be supposed, however, that gummata necessarily belong to the tertiary epoch; they may be met with even early in the disease. They occur in two forms, the superficial, forming in the skin, and the deep, in the subcutaneous tissue. The *superficial gumma* is a dull red, hardish elevation, from a small shot to a pea in size, and indolent in growth. It may either soften, and be absorbed, or ulcerate and scar. In either case some pigmentation remains round a permanent white cicatrix. The tubercles are usually multiple, but may be grouped together, so as to produce a fairly extensive red and scale-covered patch. Starting from one or several points, the gummatous process may advance over considerable tracts of skin. On one side the linearly or crescentically disposed tubercles break down, and cicatrise; on the other, the morbid process is ever being projected onward into new ground.

The *subcutaneous gumma* is more often single, and destroys more deeply than extensively. The surface is at first natural in colour, and the swelling may or may not be fixed to the skin. As it enlarges, the tension increases, occasioning a degree of œdema. It softens gradually, and conveys to the finger a deceptive sensation of fluctuation, but if incised, only a glutinous fluid escapes. When fully formed, the hue is dull red or reddish violet. Resolution may occur in it also, particularly if proper treatment is commenced in time.

The shape and appearance of the *syphilitic ulcer* is peculiar. It presents a punched-out aspect, with a thickened livid red margin. The floor is grey, and the secretion abundant in proportion to the rapidity with which it has formed. Fungations may spring from its base, and pain is commonly complained of. On the leg the resemblance to simple callous ulcers may be very close; indeed, even experienced observers have overlooked the specific element. The scar finally left is dead white, pliant, though sometimes raised; if over articulations, considerable distortion may be produced.

Gummata and their results must be diagnosed from tuberculous formations. These soon soften, the skin gets thin, and from several apertures a watery pus escapes. The ulcer is flabby, with overhanging, undermined edges, and the loose skin becoming attached, an uneven scar arises. Though occasionally lupus spreads rapidly and deeply, its progress is in general slow in comparison with syphilis; it originates early in life as a rule, its nodules are soft and semi-translucent, and the ulcers have not the excavated appearance. In lupus erythematosus the absence of ulceration, or of any sign of past or present specific disease, aid discrimination, though at times much care must be exercised before deciding. In rodent ulcer the parchment-like transformation of the skin, with the slightly raised, pearly, rolled edge, is distinctive in the early stage, while the situation—in nearly all instances on the upper half of the face—and the hard, red, granular-looking, worm-eaten ulcer, are characteristic. In elderly patients, tertiary syphilitic lesions of the face in some cases resemble the sebaceous type of carcinoma, but, almost invariably, typical scars are elsewhere to be found. Women who have become infected through the fœtus may develop gummatous lesions at a later period, without any ascertainable history of any previous cutaneous eruption. Such cases must be diagnosed on their merits.

A very obstinate affection belonging to the later period is a dry, scaly, or peeling erythema of the palm and sole, commonly unilateral. This, commencing about the roots of the fingers, tends to extend over the palm, occasionally healing in the centre, or for a time. On the sole it often leaves a space in the centre on the inner side intact, bounded by a sharply-defined curved edge. Sometimes it is more localised in the form of patches, arranged crescentically, and spreading outwards. It is possibly, as Unna has suggested, a combination of eczema and syphilis, and is distinguishable from the simple dry, scaly eczema of the palm, by the symmetry of the latter, its being worse in the centre, occurring often in gouty subjects, and giving rise to itching or burning sensations. Ringworm, affecting the palm, can be discriminated by the microscope, and dermatitis repens takes origin in an injury about the nail, from which a creeping inflammation, undermining the epidermis, steadily advances.

Syphilis may be communicated to the offspring by one or both parents, at the time of conception or during pregnancy, and the result to the fœtus is not materially different in either case. The mother remains protected since she has become syphilised either directly or through the placenta, but the child, if suckled by a healthy nurse, may impart the disease to her. If born sound in appearance, the faulty condition nearly always shows itself within the first three months. The child misses over the primary period, and so slight may be the manifestations corresponding to the secondary, that they may escape the notice of its mother or nurse. More commonly the infant wastes, assumes an oldish, wizened look, with a dingy, harsh skin, has "snuffles" or fissures about the lips. The earliest rash is erythematous, and from occurring on the buttocks, thighs, and genitals, is like the common intertrigo of these parts. It is brownish red or coppery, with a smooth, varnished surface. Combined with this, affecting other parts, is the flat papular, readily passing into condylomata in moist situations, or arranged in crescents on the face. An eruption almost peculiar to the inherited form is the bullous, a severe and early type. It has special affinity for the palms and soles, though appearing elsewhere. A bullous eruption in infants, not affecting the palms or soles, is not specific. If the child survive, all the manifestations on the skin disappear, in nearly all cases, within a year. A serpiginous ulcerative eruption, gummatous in character, may appear from the age of 5 years to puberty. In some cases it penetrates deeply, with dark pigmentation bordering the advancing edge.

There is a peculiarity with regard to the infectivity of inherited syphilis of consequence. While in the acquired disease the power of communicating it to others grows feebler the longer the lapse of time from the date of the primary symptoms, each succeeding syphilitic infant seems as capable of imparting it as the previous ones. As a rule, the disease decreases in severity in each succeeding child, but this is not invariable; it may affect consecutive children irregularly, or those born later may even suffer in a more marked degree.

Prognosis.—The prognosis of syphilis with respect to any immediate danger to life is in most cases good, in a few there are permanent traces left in the shape of indelible scars due to pustular or ulcerative eruptions in the secondary period. Still, in no case can positive freedom from tertiary manifestations be guaranteed, however skilfully or carefully treatment has been carried out, and while many of those who have passed through an attack live to a good old age, there are, on the other hand, a large number who succumb at, or soon after, middle age from complaints certainly connected with the disease from which they had suffered long years before.

Treatment.—The question of the proper treatment of syphilis has become narrowed down into that of the best modes of administering mercury and iodide of potassium, in the earlier and later stages respectively. It is true that the symptoms in the primary, and most of those in the secondary, stages will, even if untreated, in time disappear; but, in view of the persistence of tissue changes long after the eruption has ceased to be visible to the eye, it is certainly our duty to expedite the fading of the cutaneous manifestations as speedily as possible. Even those few who still hold out against the employment of mercury in syphilis affecting the skin, have recourse to it when the eye is involved, thus showing their illogical position. One has only to observe how the health improves, the anæmia and headaches vanish, and how, in nearly all cases, the eruptions rapidly pass away, under the judicious use of mercury, to be convinced of its value. In the primary stage, the chancre, if in an accessible situation, should be kept covered with a piece of pliant mercurial plaster, so as to guard it from irritation. Should this plan be inapplicable, then black wash may be substituted. Should the sore be a mixed or soft one, then iodoform one part, and boric acid three, used sparingly, forms the best application. If phagedæna sets in, rest, irrigation with a dilute, non-irritating, antiseptic—iodoform, and such surgical measures as may be needed.

Notwithstanding the evidence which Hutchinson and Jullien have furnished of the efficacy of mercury when its administration is begun as soon as the induration of the chancre affords proof that the case is one of syphilis, it seems the preferable method to delay till, from the unmistakable appearance of secondary symptoms, the diagnosis is confirmed. It is true that the early exhibition of mercury postpones, sometimes altogether prevents, the occurrence of skin eruptions, but this is not invariable, and there is no use in giving mercury unnecessarily. Every one has his own mode of giving mercury, and in any way introduced it acts in neutralising the syphilitic virus. It is most suitable in most instances to prescribe it in the form of a solution to be swallowed. One grain of corrosive sublimate and half a drachm of iodide of potassium are dissolved in 2 oz. of water. Of this, 1 drm. freely diluted is given twice a day. Sometimes half this dose is enough, or in urgent cases the dose may sometimes need to be taken three or four times a day. The teeth are to be carefully brushed, a tabloid of chlorate of potass sucked slowly twice daily, and on the appearance of any tenderness of the gums, in spite of these precautions, the drug is to be intermitted till this has disappeared, then perhaps resumed in a smaller dose. If gastric or intestinal symptoms show themselves, traceable to the medicine, the same course is to be pursued. The administration should, if possible, be continued for six months, then at intervals for another six. Smoking should be given up, and the employment of stimulants also; if such are indulged in, it ought to be only at meals, sparingly and dilute. The diet should be varied but simple, vegetables may be partaken of, but anything apt to give rise to diarrhœa must be eschewed. In the rare cases in which mercury so used disagrees, inunction should be tried before it is discarded.

The disappearance of the local symptoms is often hastened by the application of mercury directly, as in the form of black wash, dusting with calomel in condylomata, the application of weak ammoniated mercury ointment, etc. Sores on the tongue may be touched thrice a day with a solution of chromic acid, 10 grs. in 1 oz. of water, while a mouth wash containing hazeline or listerine is also useful.

Should tertiary symptoms show themselves, we possess in iodide of potassium our most efficient remedy. Some fail to yield to it, but heal when mercury is substituted for the iodide, or employed in association with it, preferably by inunction. There is no need to use large doses, 10 grs. three times a day are enough, often less may be given with advantage, but it is well to add some ammonia, in the form of the carbonate or of the aromatic spirit. Should the iodide depress, or should eruptions due to it appear, it may be necessary to discontinue it, but tolerance is established in most cases, and the remedy, when appropriate, seldom disagrees. If the gumma is entire, it commonly resolves under the iodide, but should ulcers have formed, these must be cleansed by boric-starch poultices, and dusted with iodoform. Since the iodine is excreted by the kidneys in great measure, it is important that it be ascertained that the kidneys are sound and acting well.

In inherited syphilis, inunction is immeasurably the best mode of administering mercury, and should, if at all possible, be employed. At the same time scrupulous cleanliness must be observed; the child, if not nursed by its mother, should be brought up on the bottle, on cow's milk suitably diluted with lime-water, and not on any of the patent foods or preserved milk. A wet-nurse is undesirable, since she runs a serious risk of infection.

RHINOSCLEROMA.

A rare form of new growth related to the granulomata, associated with the presence of a special bacillus, characterised by an aggregation of nodules of cartilaginous hardness, limited to the naso-pharyngeal mucous membrane and skin of the upper lip, having an extremely indolent course. It was first described by Hebra and Kaposi in 1870, and since then more than a hundred examples have been published. It occurs chiefly in Austria and South-west Russia, three instances only having been recorded in Britain. It attacks preferably poor though vigorous individuals, between the ages of 14 and 30.

Pathology.—Commencement probably takes place in the lymphatics, but the structure in the main consists of large exudation cells, with an increase in most parts of the connective tissue. Changes occur in the exudation cells, some become dropsical, others undergo a hyaline degeneration. Within the former the bacilli of v. Frisch have been found; Unna has doubts of their presence in the latter. The micro-organism is a capsule bacillus, very like the pneumococcus of Friedländer. It is also met with, embedded in glœa in the lymph spaces and vessels.

Symptoms.—The disease begins as a hard nodule within the nose or on the upper lip, painless by itself, but aching severely if squeezed. Fresh nodules or raised patches form slowly, and unite into an irregular dense smooth prominence, either not differing from the natural colour of the parts, or bright or brownish red, over which dilated vessels ramify. The glossy surface is apt to crack, and exude a sticky fluid, which dries into adherent yellowish crusts. In time it occludes the nostrils, causing much annoyance from its immobility and hardness. It exhibits no tendency to spontaneous disappearance, recurs rapidly if excised, and may continue for many years without in any way affecting the health.

Diagnosis and treatment.—This can hardly be difficult, if the peculiar situation, the stony hardness, and its slow painless growth without tendency to breaking down, are borne in mind. The sole condition resembling it is keloid, which is scarcely encountered in or near the nose. Lang obtained a certain amount of success by injecting solution of salicylic acid of a 1 or 2 per cent. strength into the growth, besides applying it in various ways externally. More recently, Stoukowenkoff claims to have cured a case, by the long-continued injection of from 1 to 8 per cent. of liq. arsenicalis.

KELOID.

A dense fibroma affecting the corium, commencing in some injury to the skin, implicating the cutis but not passing below it, but which may in process of growth advance at its periphery into healthy integument. Alibert, in 1814, was undoubtedly the first to establish the peculiar nature and clinical characters. Hebra and Kaposi cleared up various misconceptions regarding it and its supposed varieties. Hutchinson has shown that its origin can always be traced to scar tissue, and that in all its forms it manifests a tendency to spontaneous resolution.

Etiology and pathology.—Histologically, the disease in its early stage exhibits dilatation of the vessels of the corium with cell infiltration in their neighbourhood, but very soon there is found a fibroma, the bundles of connective tissue composing it running in the course of the vessels; these are compressed and obliterated, the adjacent structures are pushed aside, so as to produce a parallel-fibred cylindrical tumour, almost like tendon, enclosed by circularly arranged bundles of condensed normal skin tissue. The tension which this exerts stretches and thins out the corium and epidermis, but occasions a prominence on the surface, since it does not extend to nor involve the subcutaneous tissue. The hypertrophy of cicatrices, such as burns, is precisely similar in its essence, but the process does not advance so far; hence restitution by absorption is a simpler and more frequent result. In both, the starting-point is from the vessels.

There is but one form of keloid: the division into false and true is now given up. In its causation constitutional predisposition or proclivity is an important factor. It is more common in the dark than the white races. A hereditary tendency can sometimes be traced. Unna is of opinion that the scratching which patches of seborrhœic eczema occasion on the sternum may explain its frequency there, but scars from acne are equally common. Once initiated, the process may invade healthy adjoining skin, or may possibly infect other and remote scars. A spontaneous cure is the more likely, the more definitely it restricts itself to scar tissue. Hutchinson holds that there are forms of keloid closely allied to cancer; the isolated early nodules of non-pigmented sarcoma are very like it, but these in time break down and ulcerate, a result which rarely if ever occurs in keloid.

Symptoms.—Clinically, it may take the form of a nodule, rising abruptly and in isolated fashion from the surface: or as raised broad-ridged swellings, with irregular margins, from which claw-like spurs project laterally. Its surface is smooth and polished, of a white or pale pink colour, on which some dilated vessels may ramify. It has a firm, dense, somewhat elastic feel, is tender when pressed, or at times spontaneously painful, itchy, or the seat of a burning sensation. Perhaps its most frequent situation is over the sternum, but it may occur anywhere, least often on the face. The female sex suffer oftenest, it is seen chiefly in adult life, and does not influence the general health. Having attained a certain size, the growth becomes harder and paler, and may remain for long stationary, or very slowly increase; eventually, though possibly only after many years, resolution sets in. If excised it almost invariably recurs, and in a more aggravated form.

Treatment.—The treatment in keloid is in the main palliative. Protection from irritation is obtained by coating it with flexible collodion; if itching or burning sensations are present, 5 or 10 per cent. of ichthyol may be added. Involution has taken place in some instances after repeated electrolysis or scarification, but neither method is certainly curative.

FIBROMA.

Soft, rarely hardish tumours, due to overgrowth of the connective tissue of the deeper part of the corium, sometimes of the subcutaneous tissues, in others from the sheaths of the nerve cords.

There is still a considerable confusion as to the nature and origin of the fibrous new growths of the integument. Hutchinson classed the harder, usually solitary form with keloid as a subcutaneous variety. v. Recklinghausen showed that many instances of multiple fibromata were really neuro-fibromata, while other examples of fibroma molluscum are more properly soft nævi. Still there are cases which, whatever their exact nature, may be clinically at least described as fibromata.

Etiology and pathology.—The substance of the tumour is made up of a soft transparent connective tissue, in the centre or in early specimens loose and gelatinous, at the exterior fine and delicate, or coarse and fibrous. Unna has found peculiar granular or mast cells. The growths originate in the deepest part of the corium or beneath it, either from the connective tissue of the glands or vessels, or from the nerve sheaths. The tendency to their development is a congenital one, though they may not be conspicuous till adolescence or even late in life. Some of those affected are stunted in stature, and mentally obtuse.

Symptoms.—In the more usual form there are rounded elevations seated under the skin, which feel firm, tough, and elastic. When small they can be pushed back, like a hernia, through an aperture in the dermis, or rolled between the fingers. Their ordinary size is from a pea to a marble, but they may grow much larger. The skin investing them may be tense or loose, and either of its natural tint, or bluish, or more rarely pinkish, or plum-coloured. In general they slowly enlarge, may in time, owing to a constriction taking place at their neck, become pedunculated, or the tumour through absorption is retracted, an empty pouch of skin remaining. An exaggeration of this process gives rise to the formation of large pendulous flaps of hypertrophied integument, called *dermatolysis*. This must not be confused with a peculiar looseness and abnormal mobility of the framework of the skin, allowing it to be stretched to an unusual degree, examples of which are from time to time on exhibition as "elastic skinned curiosities." The skin of those affected with fibromata is often marked with brownish pigmentary stains, freckles, and violaceous prominences and blotches. The tumours are chiefly seated on the trunk, sometimes in great numbers, but may occur on the head and limbs, and even on the mucous surfaces. They are painless, occasioning no inconvenience save from their size or appearance.

Diagnosis and treatment.—They could only be confounded with multiple cutaneous cysts resembling the ordinary wen, or with cysticerci of the subcutaneous tissue. In either case an exploratory incision would enable an exact decision to be reached. Small ones may be treated by electrolysis, large can be cut out, but the numbers in many cases preclude interference.

MYOMA.

Firm, dull red, slowly growing tumours, which form in the true skin, may be single or grouped, cause no uneasiness at first, but subsequently may be the seat of severe paroxysmal, spontaneous pains, and are mainly composed of unstriped muscle.

Virchow seems to have been earliest to notice these tumours. Besnier, however, has given a full clinical description, on which, as also on Babes' article, Crocker has based his recent account, which comprises an abstract of all the recorded multiple cases.

Pathology.—They consist of a dense meshwork of muscular fibres running in all directions, closer in the centre, but wider at the periphery, so as to permit the entrance of blood vessels. The elastic tissue is abundant, nerves are found at the margins, but only very few within the tumour. The multiple seem to originate either from the arrectores pili, or perhaps from the muscular coat of the vessels. The other structures of the skin are solely implicated mechanically. The pain is probably due to tension or pressure of nerve endings outside the enlarging growth.

Symptoms.—They may be divided into the deep and the superficial. The former are the more common, are usually single, may be vascular and contractile, and are frequently complicated by the coexistence of a nævus. They are generally observed on the breasts or genital organs. The superficial may occur anywhere, and are arranged without regard to symmetry. The individual growths are moderate in size, seldom being larger than a pea, increasing slowly both in volume and number, tending to become aggregated into closely set patches. Their colour is mostly a brownish red, they are seated in the skin and are movable with it, their surface is smooth and natural, and their consistence firm. Sooner or later in a majority of cases they become painful, the accessions being spontaneous and paroxysmal, of varying duration, but apt to be excited by pressure or changes of temperature. In character they are benign, do not affect the glands, and do not recur if completely excised.

Diagnosis and treatment.—The diagnosis of such rare tumours is not always easy, but the characters described should facilitate it. They seem to have been confounded with xanthoma diabeticorum; but in these itching is a prominent feature, while the development is more rapid. In solid lymphangiomata there is no pain. The treatment is thorough excision, but this does not in all cases cause cessation of the pain.

VASCULAR NÆVUS.

This is a congenital over-development of the vascular tissue in the skin. It may be mainly capillary or chiefly venous. They may thus occasion a more or less extensive reddish mark, which on close examination is seen to be made up of multitudes of dilated capillaries, which can be emptied on pressure, to refill immediately on its removal, and causing no projection of the surface. Such are so common as hardly to be regarded as an abnormality, on the nape of the neck, encroaching a little on the hair margin. They constitute one form of the *port-wine mark,* so often occurring on, and occasioning so much disfigurement to the face, but met with elsewhere. The *venous nævus* is more distinctly raised above the surface, may be lobulated and compressible, if over bone may be pulsating or erectile, and is livid, purplish, or less frequently reddish in colour. They may be seen at birth, but more often it is rather the tendency to their production which is congenital, as when first noticed they may be but a pin-head in size, enlarging with the infant's growth till the bulk of a bean or walnut or much more is attained. Some disappear spontaneously, others persist, or painless ulceration takes place, with scabbing, and ultimate scarring. The cause is unexplained, but must consist in a deficiency in resistance in the fibrous meshwork of the skin; so that a species of vascular hernia occurs at one point, and the process, once initiated, extends. There is no adequate treatment, so far, for the superficial capillary nævi when fully developed, but Unna claims to have cured such by repeated painting with a 10 per cent. ichthyol collodion, provided a commencement is made in early infancy; but the prominent venous ones, if growing rapidly, or showing no sign of spontaneous involution, can be treated satisfactorily by repeated electrolysis. Needles, isolated to near their points by a coating of guttapercha, are introduced into the nævus, through the yet sound skin, at a distance of a quarter of an inch off. One is then connected with the positive and one with the negative pole, from four or six large freshly charged cells. The needle connected with the negative pole is from time to time freely moved about, so as to break up the structure. It acts as an alkaline caustic. The nævus should be frequently felt; on any sensation of its becoming dense or doughy, the action is stopped, the needles withdrawn, and the apertures closed with lint painted over with collodion. Care must be taken not to continue the electrolytic action till the growth becomes blue, else it may slough. Repetition is not advisable within a month or six weeks. As the operation is painful, an anæsthetic is necessary. If carefully done, little or no scar eventually remains.

TELANGIECTASIS.

Acquired vascular dilatations are not infrequent. One of the forms is the *spider nævus,* which is not, however, congenital, though most common in the young. It consists of a central red dot or ampulla, the aneurysmal loop of an arteriole, from which radiate minute vessels having a stellate arrangement, composed of dilated venous radicles. One or several may be seen on the bridge or tip of the nose, but such also appear on the cheeks. An injury, as a blow, is sometimes blamed; but the cause is generally unknown. In elderly persons we find on the trunk slightly elevated crimson, red, or purplish spots, from a pin-head to a pellet in size, consisting of a coil of dilated vessels. Another and rare variety has been termed *infective angioma* by Hutchinson. The disease consists in the formation of minute, bright red, vascular points and lines, like "grains of cayenne pepper" embedded in the skin. It slowly extends by the appearance of fresh outlying spots, grouped at first, but which clear up in the centre, so as to take a ring-like arrangement in some parts or cases. The skin between the rings is generally of a pinkish hue. There are thromboses, in addition to dilatation of the vascular loops, at the apices of the papillæ. It is superficial, and causes no sensations. It may originate in childhood, but in my case began in adolescence. Dilatations of the superficial venules are seen in alcoholics and dyspeptic persons at the sides of the nose, in persons with fair complexions and thin horny layer on the malar portion of the cheeks, constituting what are called "broken vessels." These are seen in association with rosacea; but, apart from it, in weather-beaten subjects, sometimes from exposure to strong sunlight. The best treatment for these conditions is electrolysis, introducing the point of the needle attached to the negative pole of a few cells, for a space of five to ten seconds, into the dilated radicle, or the central ampulla of the spider nævus.

ANGIOKERATOMA.

Dilated vessels, on which warty-looking growths develop, occur chiefly on the extremities, and in most cases preceded by recurring chilblains. The first reported case seems to have been that of Wyndham Cottle in 1877. Mibelli, Colcott Fox, Dubreuilh, Crocker, and others have related cases. Pringle has published a complete account, and Fordyce has recorded an example on the scrotum.

Pathology.—The disease commences by the dilatation of the capillaries of the papillæ; these become transformed into cavernous blood spaces, extending up into the epidermis. There is hyperkeratosis, but not much acanthosis as a secondary feature. That the recurrent stagnation in chilblains is not a necessary precursor, is shown by Fordyce's case, where numerous angiokeratomata formed on the scrotum of an old man; and by a case of my own, where a solitary one appeared on the finger of a young lady who had never had chilblains.

Symptoms.—In nearly all, the patients were or had been liable to chilblains. In course of time minute telangiectases of a pink colour, and larger and darker red, though still small vascular dilatations, appear on the dorsum of the phalanges of the hands and feet, or on the ears. A further stage is the superaddition of a degree of warty hypertrophy of the epidermis, giving rise to a composite growth which includes the vascular dilatations. In this way a transparent greyish warty formation arises, with dark red points shining through. There is no tendency to spontaneous disappearance, but, on the contrary, fresh ones may form each winter

Diagnosis and treatment.—The diagnosis, when the characters described are identified, is easy. Ordinary warts have sometimes black specks in the plush-like structure, but not the red or purple dots, with often a similar halo round the growth, seen in angiokeratoma. If single, excision is successful; but Pringle cured his by inserting the electrolytic needle into the telangiectases.

LYMPHANGIOMA.

Discrete or closely set deep-seated vesicles, or more solid nodules, in great part made up of dilatation of the lymphatics. Tilbury Fox described the first example of this rare disease in 1879, followed soon after by Hutchinson. Since then a considerable number of instances have been recorded by various observers. A careful study of the subject was made by Francis in 1893.

According to Unna, we may divide the cases into three sections—those mainly affecting the papillary body, the hypoderm, or the subcutaneous lymphatics. Only the first and second properly belong to cutaneous disorders.

The first are known as *lymphangioma circumscriptum* or *lymphangiectasis.* In a typical instance there are clusters of more or less permanent vesicles, either closely set or with single or compound ones scattered round, and having a somewhat warty aspect. The resemblance to frog spawn suggested by Crocker is apt. The vesicles themselves have tough and resistant walls, over which vascular tufts or striæ sometimes ramify, or their roof is studded with punctate dots, thromboses. If punctured, a clear liquid escapes, limpid or gelatinous, alkaline in reaction, and containing a few lymph corpuscles. In the second variety, of which but very few instances are known, that described by Kaposi as *lymphangioma tuberosum multiplex,* there is a considerable amount of fibrous tissue, giving rise to pale or dullish red nodules, rather firm and dense, not very unlike a tuberculous syphilide. There are no subjective sensations; the complaint usually commences in early life, in the first form is generally limited to one region, in the second may be widely scattered.

Since the lymphatic circulation commences with spaces, which have no wall of their own, the condition is something more than a simple varix. Noyes and Török, indeed, appear to have proved that the process consists in a new growth of lymph vessels, partly also of blood vessels, and in a transformation of these into cavities and wide canals. In the superficial form, the histological changes are almost confined to the papillary body, and the subpapillary portion of the dermis. In the deeper, more fibrous variety, there are cavernous spaces in the hypoderm of lymphatic origin. The cause is unknown. Besnier attaches importance to their association in some cases with venous nævus; the connection between venous stagnation and lymphatic stasis has been insisted on by Unna.

Electrolysis has partially succeeded in obliterating the vesicles. In a case of my own, the application on several occasions of a 50 per cent. resorcin paste, followed by a zinc and ichthyol salve, resulted in a cure.

EPITHELIAL CYSTADENOMA.

This is perhaps the best place to describe a peculiar form of new growth, of which the exact nosological position and relationships are even yet not quite definitely determined. Many names have been applied to it, and it is quite possible that at least two distinct disorders—one an adenoma of the coil ducts, a *syringadenoma;* the other, derived from some part of the prickle layer, an *acanthoma* taking on a glandular type, as Unna maintains—have been confused. The designation is that employed by Besnier, and involves least theory. Jacquet and Darier published the first case in 1887; Perry depicted it in the "International Atlas"; Török, Phillipson, Fordyce, and Brooke have specially investigated it. It presents itself as small tumours embedded in the skin, yet slightly prominent, from the size of a pin-head to that of a pea, or occasionally larger. While primarily little differing in colour from the surrounding integument, they become faintly yellowish or bluish, more rarely pinkish, as they grow. At the same time they often become shining and translucent, though solid. Nearly all contain one or several white bodies like milium. Telangiectases may ramify over the larger. They give rise to no sensation except a slight pricking. While particularly prominent and numerous on the face, they may apparently occur anywhere; but Unna thinks that those mainly located on the upper part of the trunk are to be regarded as syring-adenomata, while those grouped specially on the face are acanthomata. A well-marked hereditary element has been traced in some, but they do not crop up visibly till shortly before puberty. Histologically, they consist of prolongations of epithelium so coiled as to form masses, within which are cysts containing colloid material. The hypothesis of Török that, in accordance with Cohnheim's view, they arise from epithelial buds snared off during embryonic life from the coil ducts or lanugo hair follicles, which, dormant for a period, later acquire activity, seems a reasonable one. Their solidity separates them from hidrocystoma, but they might be confounded with adenoma sebaceum. If treatment is desired, the larger may be excised, the smaller scraped or submitted to electrolysis.

CANCER.

An epithelial new growth, presenting aggressive, infiltrating, or metastatic properties.

Etiology.—As to the cause of cancer, a disease certainly on the increase, Hutchinson's hypothesis, that a group of cell elements, freeing themselves from the central control of the organism, proceed to develop on a retrograde plan, only carries us so far. Hereditary influence is traceable in some, but age has a more marked effect. Epithelioma of the skin in any form is rare before 40. Local and frequently recurring irritation may determine its development, at least in those predisposed. It is to this that we must attribute the action of soot in chimney-sweepers', of heavy petroleum oils in shale workers', cancer. Long-continued courses of arsenic produce cancer of the palms or soles, situations otherwise seldom attacked, as a sequence to regional hyperkeratosis. Cancer is evidence of premature senility of the tissues, yet is not necessarily so, for natural senescence may be independent of cancer. Yet a weakened part, if over-stimulated, may readily take on this morbid change. It seems not at all improbable that the abuse of tea, and its substitution for tissue-building substances, combined with its influence in arresting waste, and delaying renewal, has at least some effect in promoting that increased prevalence of cancer to which allusion has been made.

Pathology.—The morbid anatomy of these different forms of cancer presents considerable variation, which cannot be fully discussed here: but it essentially consists in the presence of epithelial structures in situations where they are not encountered in healthy conditions of the tissues involved. The epithelial new growth may thus assume a cylindrical, an acinous, or a reticular arrangement; it may take on the alveolar type, in which there are, to a certain extent, isolated masses; or it may proliferate luxuriantly and fungate, in which instance it is most liable to affect the neighbouring lymphatic glands, and give rise to metastases. The cancerous epithelium undergoes secondary degenerations, one of which is that known as the formation of cell nests, actually a transformation into hyaline, taking the shape of concentrically arranged clumps. In another form of hyaline degeneration within the cells, an appearance is assumed somewhat resembling coccidia, seen particularly in that described below as Paget's disease. Softening or liquefaction occurs, so converting a part into a colloid-like mass. While the chief change is epithelial, the corium also reacts, inasmuch as a varying amount of infiltration is present, mainly of plasma with some mast cells. The infectious extension of cancer follows the lymphatics or blood vessels, the direction being possibly influenced, as Unna suggests, by chemiotactic forces.

Symptoms.—In the form of *scirrhus* this is nearly always secondary to cancer of the mamma, though it may in rare instances occur independently. It is characterised by nodules of varying size, from a pin-head to a nut, of a reddish rose or reddish brown colour, usually seated on a red or violaceous surface, often traversed by dilated vessels. They are themselves firm to the touch, but become confluent, so as to produce a well-defined induration, which may extend over greater part of the chest or abdomen, constituting the *cancer en cuirasse* of Velpeau. The lymphatic circulation is interfered with, the glands enlarge, and the neighbouring limb swells. Eventually irregular masses arise, which break down, ulcerate, fungate, and may bleed copiously. The accompanying sensations may be of the nature of stabbing pains, or only itching or burning feelings are complained of. The resulting cachexia causes marasmus, or death may be due to internal metastases. To this the term lenticular has been attached, but there is a rarer tuberous form, in which the masses are large hard lumps, which undergo similar changes.

But much more common are the *melano-carcinomata* of the skin, which have till now been generally regarded as melano-sarcomata. Unna has pretty conclusively shown that this was an error. The point of origin is usually in a flat, soft nævus, which is, as a rule, more or less pigmented. They may spring, however, from an injury apart from a mole. There is a single flat or nodular deposit, small in its inception, but reaching in time the dimensions of a nut, or larger. It is firm, sessile, smooth, or uneven on its surface, and deep brown or black. It is occasionally painful, at least if irritated, and may break down into ulceration, is apt to bleed, or exude a blackish fluid. Other similar tumours form, and systemic infection is induced through the blood current or lymphatics, with a fatal termination. The pigment favours the epithelial growth and extension, for the darker the mole the more active and malignant the process. It is the presence, too, of the pigment which, by obscuring the structure, has led to these growths being so long regarded as sarcomatous. The cancerous epithelium assumes the alveolar arrangement, the pigment, which occurs as more or less dense aggregations of granules attaching themselves particularly to the connective tissue septa, is not uniformly distributed throughout. Widely diffused metastases, to nearly every organ, have been seen in these cases.

One of the most frequent forms of cutaneous cancer is that known as *rodent ulcer*, which in all but a small minority is localised on the upper half of the face. There is a primary, smooth, mother-of-pearl-like nodule, which may long remain quiescent or but slowly enlarge. After passing in some instances through a warty stage, it flattens out, its margin has a "rolled" appearance, is reddish or pale pink, with a certain degree of semi-translucency, and when pinched up the mass feels hard. In the centre there is often a scab pretty firmly adherent, and covering a red granular slightly ulcerated surface; or, if superficial, there may be included within or between several such growths a species of soft cicatrix. Gradual as is its progress in general, its tendency is to penetrate more in the direction of depth than extension. There is more destruction than new growth. It justifies its name if left unchecked, since in time it may eat through all tissues, till, having thus destroyed greater part of the face, death from exhaustion ensues. Neither are the neighbouring glands implicated, nor do secondary deposits form.

But epithelioma somewhat different in type may occur on the skin, either on its free surface, or where mucous membrane and skin coalesce, as on the lower lip. Ill-defined nodules, invested with a degree of scaliness, become aggregated into a disc, at first movable, subsequently more or less adherent. This may break down and ulcerate, or the epithelial prolongations extending downwards, accompanied by some elongation of the papillæ, then leaving these bare, owing to the degeneration and liquefaction of the cells of the neoplasm, a cauliflower-like aspect is assumed, to which, not quite correctly, the term *papillary epithelioma* has been attached. Bleeding is readily induced in this variety. For a time the process is superficial, but the boundary of the deeper fibrous layers or fasciæ is at length passed, and a foul, fungating, deep ulcer is produced, the glands become involved, and constitutional cachexia is generated. In a fortunately rarer form the advance is more rapid, and the process is deeper seated.

The *crateriform ulcer* of Hutchinson, usually affecting the same portion of the face as rodent ulcer, may develop from that or arise as a primary growth, having more of a bossy aspect. The excavation which develops has thick and firm walls. Microscopically it is more allied to ordinary epithelioma than rodent ulcer.

A condition which, if not strictly cancerous at first, as some hold, is at least closely analogous, and paves the way for a state eventually cancerous, is that called *Paget's disease of the nipple.* Though generally limited to the nipple and areola, it may spread over a wider area, or possibly affect other regions, as the scrotum or glans penis. It usually, however, occurs in women from 40 to 60 years of age. There is a florid, intensely red, raw surface very finely granular, from which exudes a copious, clear, yellowish viscid secretion. The border is sharply defined and slightly raised, while if pinched up the fold feels distinctly indurated. Tingling, itching, or burning sensations accompany it at first, yet the health even for long suffers no deterioration. Eventually scirrhus invades the breast, and runs its ordinary course.

Diagnosis.—The diagnosis is mainly from late specific manifestations, occasionally from lupus. In syphilis the lesions are much oftener multiple than in cancer, the resulting ulcers are crescentic, with a dirty grey floor and sharp-cut edges. The brownish red primary nodules characteristic of lupus should be carefully sought for, while the history usually connotes a commencement in early life. On the face, besides epitheliomatous nodules or one or more rodent ulcers, there may be patches of red, dry, rough, scaly, or seborrhœic skin, hardly distinguishable from eczema, yet apt to develop into cancer.

Treatment.—Cancer being at first in all, throughout in some, a purely local disease, complete removal by the knife constitutes the best treatment, if at all practicable. Even if pretty extensive, healing may be obtained by

Thiersch's method of skin-grafting. Recurrence is most likely in melano-carcinoma, least in rodent ulcer. Small local recurrences may be treated successfully by scraping, followed by cauterisation with Paquelin's cautery. In widespread superficial cancer, if operation is objected to or impracticable, scraping, and the subsequent application of chromic acid fused on a probe, will for long keep the disease in check.

SARCOMA.

A tumour of the embryonic type, having its origin in the connective tissue, composed in the main of round or fusiform cells, the stroma being comparatively insignificant, but containing in some cases yellow or brown pigment granules, derived from the blood. Though the name is an old one, the conception which it now conveys is of recent date. Köbner, in 1869, seems to have first directed attention to it as affecting the skin. Kaposi, in 1870, described a remarkable variety. Perrin, Funk, Unna, and others have made careful studies of its forms.

Etiology and pathology.—Neither the nature nor the etiology of sarcoma can be said to be definitely settled. Not a frequent disease, it is met with particularly in childhood or after middle age. Pathologically, it may be said to consist of embryonic connective tissue containing cells. There are two varieties, as the component cells tend to assume a spindle-shaped or a rounded form. The small-celled sarcoma, with spherical elements, gives rise to the softest and most malignant tumour; its cells show numerous mitoses, which are absent in the spindle-celled form. Hæmorrhages also occur, and free pigment granules. The spindle-celled variety presents a very homogeneous formation. The spindles are closely packed in parallel bundles, and as they increase they cause the glandular elements of the skin to atrophy, and the elastic tissue to disappear, while the collagenous is reduced to a remnant.

Symptoms.—The earliest manifestation of sarcoma, when occurring primarily on the skin, may be a bluish brown macule, resembling a bruise or patch of chronic congestion, evolving slowly, and commonly multiple. Or it may be a tubercle or tumour, developing out of the macule, or independently of it. Such may be either of the same colour as the skin, or reddish brown or bluish red, the deeper they are situated approximating more to the latter hue. At the same time there are hypodermic tumours, which, though occasioning a prominence, do not alter the colour of the superjacent integument. They may be solitary, numerous, or in immense numbers, varying in size from a millet seed to a pea or almond, though individual examples are larger; their consistence is generally hard yet elastic, peculiarly firm should a large area be involved, but on the contrary they are sometimes soft, angiomatous, and compressible, bleeding freely if punctured. As regards its mode of development, there are three principal types—(1) That of Kaposi, which for a time localises itself on the extremities. There is brawny œdema of the hands and feet, which assume a sort of plum colour, the skin is hard and immovable, and on this the sarcomatous nodules develop. The disease progresses centripetally till the whole body may be attacked. There may be severe pain, yet the health often continues for long good. Some of the nodules may disappear, entire recovery has been known to take place, but most cases end fatally. (2) In another form the tumours form on different parts of the body, other than the extremities, and multiply gradually or rapidly. (3) A single tumour remains localised for a certain period, then rapid generalisation takes place.

Diagnosis and prognosis.—This is not in all cases easy. It must be carefully separated from syphilitic gummata, mycosis fungoides, cysticerci affecting the skin and subcutaneous tissue, the nodules of lupus and leprosy, and perhaps, in Kaposi's form, from Raynaud's disease. The prognosis is always grave, for, though spontaneous disappearance of the tumours has occurred, and even cure has resulted, this is rare. In view of this the favourable effects of the subcutaneous injection of liquor arsenicalis in the treatment, recorded by Köbner and Shattock, lose a certain amount of their value.

MYCOSIS FUNGOIDES.

A rare disease of the skin, characterised by the formation of painless tumours, somewhat resembling tomatoes, preceded in many cases by very itchy eruptions, erythematous, lichenous, or eczematous in type. It was first described by Alibert in 1832, who attached the term mycosis, from the similarity of the tumours to mushrooms, not from any supposed parasitic etiology; the affix "fungoides" refers to the tendency these have to burst open. Vidal, Brocq, Payne, Tilden, Besnier, and many others have rendered our acquaintance with its features very complete.

Etiology.—As to the cause, we are completely in the dark. It is most frequent in men, and particularly in late adult life. No evidence of contagiousness has been adduced, and the antecedents of those attacked, as of the relatives, has generally been good. Some regard it as allied to sarcoma, others to lymphadenoma, but it seems independent of either.

Pathology.—When the growths are examined they are found to consist in the main of cells of the lymphoid type, contained in a delicate reticulum. Individually these vary in shape, but are frequently oval, with one or more nuclei. Their protoplasm stains rather badly, so that their outline is ill-defined. The tumour is watery in consistence. The epidermis is thinned. Phillipson has examined the early patches, and finds that these present anatomically the same characters. The commencement seems to be in the neighbourhood of the blood vessels, the origin being from the connective tissue; but according to Unna the early cellular infiltration shows a saturation with granular protoplasmic particles. No organisms having any significance have been met with, though in the later stages such accidental visitors are plentiful.

Symptoms.—In one class of cases three fairly distinct stages can be recognised. In the earliest, a superficial and extensive eruption appears. This often is much like patches of dry eczema, or it is a widely diffused erythema, perhaps with wheals. There is no impairment of health, though usually severe itching, less often burning sensations, are complained of. This stage may last many years. By degrees, however, the skin on the affected parts becomes thicker, it acquires the aspect of a patch of long-standing lichen planus, yet on these bullæ may arise. This constitutes the second period. Sooner or later the third, that of tumours, is reached. Either on one of the infiltrated areas, or on sound skin, a nodular elevation forms, frequently quite suddenly. This is primarily firm, later as it enlarges it softens. Commonly such are brightish red, but they may be yellowish white, or even plum-coloured. Their surface is smooth and glossy, the epidermic covering being thin and tightly stretched. Though no pain accompanies them, the pruritus persists, so that they may be torn, or, spontaneously rupturing, a gummy or bloody fluid exudes from their succulent mass, and dries into crusts and scabs. By increase in numbers they coalesce so as to cover wide areas, involving in general the head and upper part of the trunk before the lower limbs are invaded. Crater-shaped, often deep, ulcers may form. The lymphatic glands may enlarge. A nauseous, peculiar, and offensive odour is exhaled. While some of the growths continue throughout, others shrivel up and disappear, leaving little or no trace. Eventually the health, hitherto good, fails; emaciation, feverishness, septicæmia, and death from exhaustion, sometimes with colliquative diarrhœa, occurs.

In another class of cases the stage of tumours is reached at once, no diffused eruption preceding their evolution. Itching is not so marked, and purplish or reddish blue macules are seen apart from, or as an areola round, the growths, or left behind by some which have become absorbed.

Diagnosis.—In the early stage, where there are superficial eruptions, the diagnosis is difficult unless an examination of an excised portion of affected skin be made. Persistent itchiness, with rebelliousness to treatment, might arouse suspicion. The fully-developed disease is not unlike tuberculous leprosy; but there is no anæsthesia nor destructive ulceration.

Treatment.—As to treatment, arsenic, while it has apparently benefitted some examples, has aggravated others. Bazin's first case entirely and permanently recovered after an attack of erysipelas; hence Pringle suggests further trial of serum therapeutics, though so far the results have been negative. Any gangrenous tumour should be excised, as Hallopeau has shown that such may be the cause of hectic fever.

SENSORY DISTURBANCES.

Following the view of Crocker, it seems the simplest plan to regard as neuroses of the skin only those disorders of innervation in which the changes are functional and the symptoms subjective, the secondary effects being the result of accidental circumstances, rather than to adopt the wider conception held by many, of including within them all those diseases whose pathogenesis has been proved or suspected to lie in some alteration in the nervous centres.

HYPERÆSTHESIA.

Exaltation of sensibility of the skin is nearly always symptomatic of some deeper-seated source of irritation, not perhaps readily discoverable. It is also a manifestation of hysteria, and in the recognition of this element will be found the key to its cure.

DERMATALGIA.

By this is understood cutaneous pain, independent of any appreciable alteration in the integument or nervous system. The condition is rare, and distinguishable from ordinary neuralgia by its superficial character. It is paroxysmal, usually local, and principally met with on the scalp. Unna locates it in the hair follicles, and caused by cramp-like spasm of the muscular structures in these. The fact of its being spontaneous separates it from hyperæsthesia.

ERYTHROMELALGIA.

Under this term Weir Mitchel has described a chronic ailment in which a part or parts, commonly one or more extremities, suffer from pain, flushing, and local fever, made far worse if the limbs hang. It appears to be due to a diffuse or terminal neuritis, related, according to Personali, to syphilis. It may occur in combination with Raynaud's disease, but is separable from it.

PRURITUS.

This perversion, of which itching is the sole direct symptom, is by far the most important. It may be either general or restricted to certain areas.

Symptoms.—*General pruritus* is specially encountered in advanced life as *pruritus senilis*, and is then one of the consequences of that atrophy of the skin almost inseparable from age. Of all the changes in the skin as years advance, that which has the chief influence in initiating pruritus is dryness, a cessation more or less complete of the insensible perspiration which does so much to preserve the integument in health. The skin of the aged—a relative term not always commensurate with the years lived—is pale, thin, silkily-smooth, or wrinkled, discoloured, and rough from accumulation of the horny cells. Complaint is made of paroxysms of itching on undressing, or when warm in bed, or after a diffusible stimulant has been partaken of. The scratching resorted to may or may not leave visible traces; it is often indulged in till pain has replaced the intense pruritus. In severe cases the sleeplessness and wearing effects of the peripheral irritation react on the general health, even inducing impairment of the mental powers. In some, bilious attacks, sudden accessions of acute gastric or duodenal catarrh, occur at indefinite intervals. These bring it into relation with the pruritus so common in protracted jaundice.

General pruritus may also be one of the subjective symptoms met with within the cycle of the "change of life," an expression of the disturbance to the economy occasioned by the arrest of a rhythmical function. It is then sometimes associated with a degree of mental aberration, either hysterical or bordering on actual insanity. It often assumes the form of formication, or the sensation as if myriads of insects were crawling over, stinging or biting the skin. This "parasitophobia," as Perrin has designated it, may be secondary, when it is a sequela of a pre-existing though cured scabies or pediculosis; or primary, if it develops in those who have never had such ailments, though possibly the idea has been started through their having mixed with those who have.

What is called *pruritus hiemalis*, described by Hutchinson, Duhring, and Payne among others, is a third form. This is an itchiness of the skin in cold weather, especially when the air is keen, dry, and frosty. It arises when undressing to go to bed, or in a warm room if the external temperature is low. "Change of temperature rather than absolute cold is the exciting cause, while dryness of the air is an important factor."

The commonest form of local pruritus is that known as *pruritus ani.* This is not limited wholly to the seat, but extends to the perineum. Little or no alteration may be visible, or there may be slight secondary redness, thickening, or cracks. The sufferers are mostly sedentary men, whose occupation is monotonous, worrying, or disquieting. In some the presence of piles or varicosity of the lower hæmorrhoidal veins can be shown, or there is urethral or prostatic disorder, or ascarides are the source of irritation; but the patient may be otherwise apparently healthy, though he may live freely or smoke too much.

Another variety is *pruritus genitalium.* That affecting the vulva is the most frequent, and in all such the existence of saccharine urine should be sought for. It may, however, be climacteric, or associated with a varicose condition of the labial veins. Leucorrhœa may be the cause, or merely the accumulation and rancidity of the secretions irritating the clitoris, whence the itching radiates. It may attack the scrotum and perineum in men, as a result of varicocele, or the meatus urinarius in stone or diabetes.

A local form of pruritus hiemalis is that occurring between and under the smaller toes when the stockings are removed at night, or prior to this in the evening after dinner. There is also a *pruritus narium* met with in the gouty, either with a dryness of the lining membrane or a constant dripping of a clear fluid.

Diagnosis.—In the diagnosis of general pruritus, pediculosis and urticaria must be excluded. Evidence of the insects may be found in the clothes, and a history of the eruption of wheals may be learned on inquiry.

Treatment.—In the management of pruritus we must bear in mind that it is a superficial phenomenon, which, in accordance with the view of Bronson, may be regarded as having its essential seat in the epidermis. "It is always associated with a presentiment to consciousness, as though a foreign body were in contact with the surface." Unna explains the perversion of sensation as occasioned by a disproportion between the pressure of the tissue juices and the resistance exerted by the horny layer. Our efforts, therefore, should be directed to remove all sources of irritation, and to prevent recourse being had to the natural mode of relief, namely, scratching. This, though affording relief for the time, partly by substituting pain or smarting for itching, lowers the vitality of the skin, and increases its subsequent sensitiveness. Actual restraint is seldom applicable, though something may be done in the way of preventing the hands having access to the itchy part during sleep. The underclothing ought to be either of cotton, linen, or silk; woollen articles are seldom tolerated. Such may, however, be worn over those mentioned. For washing, some well-made superfatted soap and soft tepid water must be used, the surface being dried with a linen towel, without friction. Warm alkaline sulphur baths, followed by inunction with a bland salicylated oil, such as oleum declina, containing 5 to 10 grs. of salicylic acid in the ounce, or vaseline with a like amount, will be found beneficial in all forms. We may in like manner employ menthol dissolved in oil, in much the same proportion. This acts by substituting the sensation of coldness for that of itching. It may likewise be used as a 5 per cent. superfatted soap. Pruritus senilis may often be permanently cured by injecting from $\frac{1}{10}$ to $\frac{1}{3}$

of a gr. of a salt of pilocarpine subcutaneously at night. The action of the pilocarpine as a diaphoretic should be kept up for an hour by imbibing hot water freely, and its use should, to be effective, be continued for at least ten days or a fortnight. In all forms of pruritus the avoidance of sudden changes of temperature should be aimed at as much as possible, as also the use of alcohol or articles of food or drink likely to flush the skin. In the local varieties, painting with a solution of nitrate of silver in sweet spirits of nitre, 10 to 15 grs. in the ounce, is of much use, employed occasionally. Carbolic acid dissolved in oil or water, 10 gr. to the ounce, is also a reliable antipruritic, in such forms; and much the same may be said of dilute solutions of tar, as 3 per cent. of liq. carbonis detergens in water, or as an ointment with oxide of zinc in cold cream. Douching with water as hot as can be borne, and then dusting freely with talc containing 5 or 10 per cent. of boric acid, is also valuable.

ANÆSTHESIA.

This is a rare form of disturbance of sensibility, and is mostly encountered in hysterical conditions, or in leprosy.

VEGETABLE PARASITIC DISEASES.

There are many of the cutaneous disorders already described which have been proved to be occasioned by low forms of vegetable life, in some of which the suspicion is strong, though yet unconfirmed, while others are apparently independent of any such agency. These depend on the presence of organisms belonging to the schizomycetes, fission fungi, bacteria, or micrococci. But there is a class of diseases which arise from the reactive irritation of the skin to the invasion of members of the hyphomycetes, or branching fungi, in which the cell or spore or short filament has the property of elongation, again in most instances to break up into innumerable fragments, each of which can propagate the fungus.

FAVUS.

A contagious disease, due to the *Achorion schönleinii*, exhibiting pale yellow, cup-shaped, friable crusts, usually perforated by a hair if on parts where such are present. The term seems to have been used till comparatively recent times merely to denote honey-like crusts, not the comb-like arrangement of the favi themselves. Its parasitic nature was discovered by Schönlein in 1839, and two years later Gruby accurately described the fungus. Latterly discussion has arisen as to the unity or multiplicity of the parasite, a point not yet conclusively settled.

Etiology and pathology.—The favus scutulum, detached from the depression in the corium in which it is seated, is made up of branching mycelium, oval and round spores, with granular débris, containing various micro-organisms and pus corpuscles. The individual segments of the mycelium are shorter than in trichophyton; it runs lengthwise through the hair, interfering with its transparency but not dissecting its fibrous structure as the trichophyton does. Hence the hair, though lustreless, is not nearly so brittle. If a drop of liq. potassæ be placed on a hair attacked by the achorion, a cover-glass laid on and immediately after examined by the microscope with a power of 250 diameters or more, the liquid can be seen to flow through the mycelial tubes, driving the air before it. This is a good method of discriminating between favus and some forms of trichophyton with resistent mycelium. The botanical position of the achorion has not yet been determined. Sabrazès has shown that in dogs and fowls the fungus may belong to a different species from that affecting man, but though the fungus of human favus is a distinct one, which gives rise only to favus, it is still *sub judice* whether there is but one species or not. So far the clinical appearances do not correspond with the varieties of cultures obtained.

Symptoms.—Favus is chiefly met with on the scalp, and its earliest appearance is that of a minute, sulphur-yellow speck, at the mouth of a hair follicle. This as it enlarges acquires a depression in the centre while it rises at the periphery. The yellow crust finally attains the dimensions of a split pea, the older central portion being lighter in hue than the marginal. While individually roundish, and having their seat in the epidermis, when they become aggregated, pressure imparts a hexagonal contour. The masses of crust may pretty nearly cover the scalp, or form more or less isolated groups, partially enclosing bald places where the disease has died out, and set in healthy areas. Throughout its whole course it is accompanied by erythematous redness, which may proceed to other more complicated inflammatory changes. Pediculi commonly swarm in the tangled mass of withered hair which grows irregularly over the thick crusts, and a peculiar mousy odour is exhaled. It is difficult to determine how far the itchiness complained of is due to favus alone.

It may occur on the trunk independently, wide areas being affected, but in most cases this is an extension from the scalp. The same dry, yellow, crumbly, though tenacious crusts characterise it, but in addition red scaly rings are found on the trunk or limbs. It may also insinuate itself under the nail, and appears as a yellow spot beneath the nail-substance, or raising it up and protruding from the free margin in front.

While chiefly seen in the young, it is eminently chronic, may even last a lifetime, or only die out when all the hair follicles have been destroyed. Many of those suffering from it are pale and lymphatic, the glands near the diseased areas are enlarged, and some hold that it exerts a deleterious effect on the general health. Though probably on the decrease, it is fairly common in Scotland while rare in London, it is seen in rural districts and in urban indiscriminately, hence it has been called the tinea of the country as opposed to ringworm, which has been termed that of towns.

Diagnosis.—The aid of the microscope should be invoked in all doubtful cases. Eczema in the strumous may occasion bald areas, as may also destructive folliculitis and lupus erythematosus.

Treatment.—In the treatment of favus the masses of crusts are first to be thoroughly softened with oil, to which some paraffin may be added in the event of pediculi being present. By poulticing and washing with green soap and warm water, the scalp can soon be cleansed, and made to assume a red and shining aspect. The hair should be cut very short. Systematic epilation must then be practised, commencing at one point and advancing steadily onwards till all the diseased hairs have been removed, and a margin of at least a quarter of an inch of apparently healthy ones beyond. The epilation may be rendered less painful if simple oil or oil of cade, with 4 per cent. of carbolic acid incorporated with it, be rubbed in to the part from which the hairs are about to be removed. Only a small part can be so treated daily. After epilation the scalp is again washed, dried, and an ointment or vaseline starch paste containing 10 per cent. of resorcin applied. It is difficult if not impossible to cure favus without epilation, but Pirogoff states that the application of the following pomade is successful:—℞ Sulphuris præcip., 15·0; potassii carbonatis, 4·0; picis liq., tinct. iodi, āā 50·0; adipis, 100·0—*M.* This is spread on cotton and applied to the cleansed patches, till it occasions an abundant desquamation. Then an ointment of zinc and salicylic acid is used. He states he has found this to occasion such loosening of the hairs that epilation is unnecessary, and cure is rapid. Removal of the crusts and application of the resorcin ointment is sufficient to cure if on the trunk. Tonics and good diet are called for. The X-rays have been employed to cause loosening of the hairs, and their more ready extraction. This result appears to follow more certainly if the special dermatitis is produced, but as this in turn may be succeeded by ulcers and subsequent scarring, the possibility should be explained to the patient. It is conceivable that this plan may prove valuable, when the experience of the technique will enable us to gauge the duration of exposure and energy required.

TRICHOPHYTOSIS.

Syn., Ringworm.

A parasitic disease affecting the scalp of children, the hairy parts of the face in adult men, the general surface, more rarely the nails, at all ages, produced by the growth of various species of the trichophyton. The parasitic nature of ringworm was established by Gruby and Malmsten about 1842. M'Call Anderson, Thin, and Alder-Smith contributed to our knowledge, but fresh interest has been infused into the subject by the observations of Sabourand, who showed that there are at least two or three species of the fungus. His views have been in the main confirmed and extended by Leslie Roberts, Adamson, Fox, and Blaxall; while Morris has introduced a simple stain.

Etiology and pathology.—In general terms the parasite consists of mycelial threads and "spores," or, more properly, independent fragments of mycelium. In this country the most common species is that called by Sabouraud microsporon, identified with the *M. audouini* of Gruby, but which some hold is also a trichophyton. It is found in at least 95 per cent. of all cases of ringworm of the scalp in children. The spores are small, 3 μ to 4 μ in diameter. These form a sheath round the hair, which is itself filled with delicate mycelial threads. There is likewise a large-spored fungus, which may affect the scalp and general surface. It consists of mycelial filaments or sporulated threads, packed side by side within the whole stump. It affects solely the interior of the hair, hence it is called *Trichophyton megalosporon endothrix.* It occasions circinate lesions of the skin. It is rare in Britain, but less so in France. Another variety is *Trichophyton megalosporon endo-ectothrix.* This gives rise to tinea barbæ and ringworm of the nails, very seldom ringworm of the scalp.

In the hair the mycelium is straight like that of favus. The spores are in most cases large in both the latter forms, varying, however, from 4 μ to 12 μ in diameter. One does not meet with an intermixture of these forms; each case presents only one and the same form, and so do all cases derived from it by communication. Probably the best way of examining suspected hairs is to place them for a few minutes in liq. potassæ, then transfer them to distilled water in a watch-glass, then mount in glycerin. In this way the fungus is beautifully distinct, and the size of the spores can be readily estimated. If scales from the surface are to be examined, a drop of liq. potassæ is placed on, then a cover-glass, and in from five to ten minutes the mycelial threads, which always predominate in epithelial scales, become clearly visible. The fungus is closely allied to the moulds. It is probably a saprophyte which has become parasitic. The small-spored variety seems of human origin, some at least of the large-spored are derived from animals. The results obtained by cultivations are somewhat confusing, as different media give rise to marked variations in the cultures.

The origin of body ringworm is often difficult to trace; the washerman in India is blamed as the medium of communication; the laundry in cooler climates may sometimes be at fault. Direct or mediate contagion by caps or brushes, accounts for epidemics in children at school or classes. Shaving generally, sometimes contact with cattle, produces ringworm of the beard. Kerion is occasionally due to irritant applications, more frequently is a question of soil.

Symptoms.—Clinically the simplest type of ringworm is that known as *tinea circinata*, seen on smooth surfaces or on those invested with downy hairs. This begins as a rose-red spot, which soon becomes a red, slightly scaly patch, oval or round, and either uniform all over, or merely a red crescent or ring bearing some easily dislodged branny flakes. Such may appear on a part covered with clothes, or on the chin or hand. In the latter situation the margin may be vesicular. On the palm or sole the commencement may in like manner be a blister, which enlarges, bursts, and exposes a red surface denuded of horny epidermis, but presenting an edge of undermined and thickened epidermis. In all these situations there are as a rule only one or two, seldom many, areas of disease. On the scrotum and adjoining parts of the inner aspect of the thigh, a compound of ringworm and intertrigo is seen, especially in warm countries. The margin of the patch, which may be many inches in diameter, is red, raised, and crescentic. The centre is paler, fawn-coloured, sometimes moist, at others scaly. It is a peculiarly obstinate form. In all a degree of itchiness precedes or accompanies the ailment; in the last variety it is especially aggressive and troublesome.

On the scalp in children there is ordinarily seen one or more circular patches, more or less completely covered with white mealy scales, and withered and dry hairs, most of which are irregularly broken off at about a line and a half from their point of exit from the skin. Sometimes, particularly where the complaint has lasted long without treatment, nearly the whole head may be thickly covered with whitish scales, while the hair, though thin, grows a fair length, but underneath this numerous broken hairs can be found. This is the type seen in connection with the small spored fungus. In that due to the large-spored fungus the lesions are more scattered and the scaliness less. There is a marked proportion of dark swollen stumps, broken off very short. In all cases the epidermis is affected before the hair. A peculiar phase is that called *kerion*. On one or more places on the scalp the skin is swollen up like a watch-glass, the surface is red, tender, moist, and either smooth or crusted. The follicles gape, exude a gummy discharge, and the hairs are loose or have fallen out. This sometimes occurs with the small-spored, more often with the large-spored fungus. It is possibly a mixed infection with pyogenic organisms. Much resembling kerion in appearance, but developing on glabrous surfaces, as the backs of the hands, the limbs, neck, or nates, not on the palms or soles, are rounded, raised, uneven reddish patches, not unlike a macaroon in shape. These are studded with numerous apertures from which pus can be squeezed, corresponding in general to hair follicles, with crusts, or unruptured pustules. The hairs have usually fallen out. The disease commences as a small boil, these multiply and coalesce. Itching and sensations of heat accompany it. There are not often many patches. The condition was originally described by Leloir as *conglomerate pustular perifolliculitis*, and he regarded it as of microbic origin. Hartzell traced its connection with the trichophyton, and Sabouraud has confirmed this. The source is apparently equine, and the variety *ectothrix*.

On the bearded part of the face there may be nothing more than one or several reddish, dry, scaly circular patches in the whiskers, on the chin or neck, rarely on the upper lip. The hairs may appear somewhat lustreless, but are not markedly involved. The disease may proceed further, the surface in parts looking as if it had been scalded or blistered. A yellowish serosity exudes abundantly, concreting into thick crusts. The hairs are more or less diseased, partly loosened. In many instances a third grade is reached; rounded, uneven, reddened tuberculous formations are produced. The hairs are loose or have fallen, and a viscid fluid is poured out. The condition closely resembles kerion. In these last cases, pain of a throbbing character is complained of in addition to itching.

The nails appear to be very seldom attacked, and only secondarily to ringworm of the back of the hand, or as an infection from the scalp, but as the complaint persists for long after the cure has been effected elsewhere, it is possible that instances have been erroneously regarded as eczema or psoriasis of the matrix or bed of the nail. The first change is the occurrence of white lines or points under the nail; this becomes unequal, is thickened, more convex, loses its transparency, and finally falls off.

Diagnosis.—In the diagnosis of ringworm of the body, patches of seborrhœic eczema, usually encountered on the chest or back, and pityriasis rosea, look most like it. But these are more extensively distributed than ringworm, and the microscope does not reveal the mycelium of the trichophyton. Mild types of psoriasis can generally be excluded by examining the knees, elbows, and scalp. A recurrent syphilide assuming an arrangement in rings somewhat simulates it, there are few or no scales, some of the lesions are crescentic, and there is no itching. A disseminate ringworm of the scalp is frequently regarded as seborrhœa. If a little chloroform is sopped on, and after evaporation the part inspected with a lens, if the case is one of ringworm, all the broken hairs assume a mealy whiteness, seen in no other condition. Some of these should then be removed, and examined microscopically. Sycosis due to pyogenic organisms differs from tinea barbæ, inasmuch as the hairs are not much loosened, and if attempts at extraction are made, pain is experienced. It is most common in those who do not shave, tinea barbæ is the reverse. A late tuberculous, ulcerating, or vegetating syphilide usually shows horseshoe-shaped ulcers at some point, the hairs may have fallen out, but a cicatrix replaces them.

Prognosis.—This is good as regards ultimate cure, though the disease is undoubtedly often obstinate. According to Sabouraud, ringworm of the scalp due to the small-spored fungus disappears spontaneously at or about puberty, but in the case of the large-spored variety this is not nearly so absolute. It may persist during adolescence. We rarely meet with ringworm of the scalp in adults.

Treatment.—The treatment of ringworm of the trunk or limbs is generally simple. Washing the affected parts twice daily with a superfatted potash soap, or failing this a similar good hard one, and warm water, and the subsequent application of an ointment of precipitated sulphur 1 drm.; ammoniated mercury ½ drm., and lanoline 1 oz., will almost with certainty cure in a week. That affecting the scrotum and thigh is more obstinate. The most effectual remedy seems to be freshly prepared sulphurous acid sopped on three times a day, the surface being at the same time kept well powdered with boric acid 10 parts and talc 90. From the impossibility of getting any remedy to penetrate to the bottom of the hair follicle, so as to act as an efficient parasiticide, Sabouraud holds that ringworm of the scalp is incurable by the means at our disposal. Though tedious, the cure can be accomplished. The hair, if not kept shaved—the best plan—must at least be cropped as short as scissors can cut it, and this must be continued till a cure is attained. It must be washed twice daily with superfatted potash soap and warm water, and the sulphur and ammoniated mercury ointment, mentioned above, well rubbed in. Another useful salve is composed of oleate of copper, ½ drm. to 1 drm. in 1 oz. of lanoline. Or 10 grs. to ½ drm. of salicylic acid, may replace part of the ammoniated mercury. In very chronic and limited cases in children over 10, the croton oil treatment of Alder-Smith may be cautiously tried. The patches are painted at intervals of a few days with pure croton oil, and over this a linseed poultice is constantly applied. A degree of pustulation is set up, and the hairs are loosened and fall out, to be replaced by healthy ones. Kerion is best treated by continuous poulticing with boric starch poultices. This, too, constitutes the best preliminary treatment in ringworm of the beard. When the surface has thus been cleansed, the superfatted soap and the sulphur and ammoniated mercury ointment, combined with epilation of all loose hairs, is commonly rapidly effectual. If the nails are attacked, soaking in alkaline solutions, scraping down, and painting with linimentum iodi seems the best mode.

TINEA IMBRICATA.

A superficial parasitic affection of the epidermis, characterised by concentric rings consisting of dry flakes attached at their periphery, occurring in warm, moist localities in the Pacific Ocean. According to Patrick Manson, who has given the most complete account, it was recognised by Dampier in 1729, Fox in America gave the first medical description, but Turner, Königer, and Macgregor have also reported on it.

Etiology and pathology.—Moistened with liq. potassæ, and examined, the scales show a most luxuriant growth of a fungus resembling trichophyton, but in prodigious abundance. Unna regards it as a saprophyte, and places it in juxtaposition to the microsporon furfur, and it must be admitted that it has many features in common with pityriasis versicolor.

The home of the disease is in the Malay Archipelago; from thence it has spread east and south to many of the islands of the Pacific and into China. Very high temperatures or conversely dry cold weather are inimical to it.

Symptoms.—Manson repeatedly inoculated it, and states that it has an incubation stage of about ten days; then a minute, slightly elevated brown spot appears. This increases, the epidermis being undermined till it ruptures in the centre, becoming separated from the underlying part as a flake. The ring enlarges still further, another brown spot forms in the middle, and the same process is renewed, the original ring having in the meantime extended. A series of concentric rings are thus produced, new foci develop, till the whole body is involved, with exception of the palms and soles, and generally the scalp. Should one system of circles infringe on another, the regularity of the pattern is disturbed. "The arrangement is something like the rings of light and dark surface on watered silk." The free border of the flake is towards the centre of the circle to which it belongs. The brown colour is due to the tint of the fungus, and if the skin is very dark the affected parts may look lighter. It occasions no redness or trace of inflammation, does not apparently affect the hair even of the scalp, and only itches in warm, damp weather.

Treatment.—This consists in applying linimentum iodi of double the usual strength to the parts, piecemeal, so as not to occasion too great irritation. At the same time the clothes must be burnt or boiled.

PITYRIASIS VERSICOLOR.

A superficial affection almost wholly confined to the covered parts, characterised by the appearance of fawn-yellow or pale brownish areas, which on being scraped give off a fine bran-like scaliness, due to the vegetation in the horny layer of the microsporon furfur.

The complaint was described by Sennert nearly three hundred years ago, but the name was attached to it by Willan. The fungus which gives rise to it was discovered by Eichstedt in 1846. Many writers have confused it with chloasma.

Etiology and pathology.—The fungus is a saprophyte, and, according to Unna, only vegetates in the middle layer of the horny portion of the epidermis. It consists of winding and interlacing mycelial filaments, including, within the meshes so formed, groups of rounded spores, differing a little in size individually, but rather less than a blood corpuscle. The alterations produced are mainly mechanical, though the slight inflammatory phenomena occasionally observed may be due to a feeble toxine. A few scales placed on a slide and moistened with liq. potassæ, show unmistakably the peculiar arrangement of the cryptogam.

Symptoms.—The eruption special to the disease consists of spots and larger areas, varying from a pale fawn-yellow in fair persons to a dark brown even in the swarthy. The surface is composed of branny scales, easily loosened by such measures as friction with the nail. The distribution is wonderfully symmetrical, the smaller disconnected spots being situated beyond the margins of the larger. In most cases it is confined to the chest, but may extend to the limbs so far as covered. Itching though sometimes complained of, is not a prominent symptom.

Though contagious, it is only so to a slight extent. Those who perspire freely are most subject to it, but regular daily ablutions have no influence in freeing from it. It is very rare in childhood, and disappears as the skin becomes drier after middle life. It is much more frequent in males.

Diagnosis.—It has been mistaken for a syphilide, for seborrhœic eczema, and for pityriasis rosea. From all these the microscope provides a certain means of determination.

Treatment.—This is simple and usually satisfactory. The skin should first be well washed with green soap and warm water, to free it from greasiness. After drying, the surface should be sponged with vinegar, and, while still wet, with a solution of hyposulphite of soda in water, of the strength of 1 drm. to the ounce, with a little glycerin. This ought to be repeated nightly for a week or ten days. Afterwards, to prevent recurrence, the use of a superfatted resorcin and salicylic soap, with warm water twice a week, for a time, to thin down the surface of the horny layer, is advantageous.

ERYTHRASMA.

A brownish or reddish brown patch, desquamating but little, occurring on the inner side of the thigh, lasting a long time, and due to the *Microsporon minutissimum;* was so named by v. Bärensprung. The parasite was described by Burchardt. Payne, Bizzozero, and Unna have investigated it.

It is said to be fairly common, but the complaint attracts little attention, since it scarcely occasions annoyance by itching, and the fungus needs careful staining, preferably by Gram's method, and a power of at least 700 diameters. There are septate filaments and numerous fine, round, or angular spores. It comes nearer to pityriasis versicolor than to ringworm. The fungus is a saprophyte which flourishes in the upper layers of the epidermis. It has been regarded as an intertrigo, but this is only an occasional superaddition. The treatment consists in the application of a dilute citrine ointment containing a little creasote.

ACTINOMYCOSIS OF THE SKIN.

A parasitic affection due to the ray fungus, producing, when it attacks the skin, tumour-like growths, which soften, suppurate, and give rise to sinuses. Bollinger in 1877 identified the micro-organism, though Langenbeck had detected it and recognised its fungoid nature so early as 1845. Its existence in cattle had been known before Israel proved its occurrence in man. Neumann, Darier and Gautier, and Pringle have illustrated the various cutaneous types.

Etiology.—The form in which the parasite is visible to the naked eye is that of opaque granules, of a sulphur or paler yellow tint, about the size of a pin-head. These in greater or smaller numbers float in a sanguineous or purulent fluid, discharged from the liquefied tumours. Crushed under a cover-glass, they can be seen to consist of a felted arrangement of straight or winding fibres, assuming a radiating plan with club-shaped terminals. It is best demonstrated by Gram's method.

The ray fungus vegetates on various members of the family of the graminaceæ, as barley, wheat, and rye, yet cultivations from such sources have not so far reproduced the disease. Still it appears certain that this is in some way the origin, and actinomycosis is pretty frequent in cattle. No case of direct transmission from animals to man has been recorded, but it seems communicable, though apparently with difficulty. It has been supposed to gain access by way of a decayed tooth, by swallowing uncooked grain, or by puncture by barley awns. It is probable, as Foulerton has shown, that there are various mould fungi, to be classed as species of streptothrix, capable of producing symptoms which have hitherto been regarded generally as actinomycosis. It is twice more frequent in men than women.

The skin in man is seldom involved, except secondarily, from some deeper source.

Symptoms.—The period of incubation may be a prolonged one. On becoming apparent there is first perceived a small roundish or oval prominence, hard as wood, seated subcutaneously. This enlarges, approaches the surface, acquires a red or violaceous colour, grows soft and fluctuating, and may finally burst, forming an ulcer with sinuous fistulæ. Secondary tumours develop progressively in the neighbourhood, and follow a like course. Thus a reddish violet patch is produced, uneven, indurated, apparently adherent to the subjacent parts, and not unlike a collection of scrofulo-tuberculous gummata. If on the cheek, one of the more common situations, it seems attached to the periosteum of the malar bones or jaw. Pain is at times severe, at others absent, or only evoked by pressure. The lymphatic glands are seldom implicated. The aspect of the growths in Pringle's case was somewhat different. They consisted of livid, fleshy, sarcomatous-looking outgrowths of mottled purplish red and yellow colour. To touch they were soft, pulpy, and fluctuating, though not tender. The integument over them was thin, stretched, with dilatation of the blood vessels. From cupped openings much clear, sticky fluid constantly exuded, while the retained purulent fluid contained granular specks in which were the actinomyces. The growths were principally situated on the right flank and back, and the source was in the lung. The skin from which they sprang was hard, brawny, and tender, and of a deep purplish brown colour.

Prognosis.—However introduced, the disease advances by gradual invasion, while its progress is chronic, and, unless checked, it in many cases eventually, if slowly, causes death by exhaustion.

Treatment.—This consists in opening up the sinuses and foci of suppuration, scraping them out thoroughly, and administering large doses of iodide of potassium—45 to 60 grs. daily—a drug which has a specific action in curing the disease.

BLASTOMYCETIC DERMATITIS.

A destructive disease of the skin, due to the proliferation of an organism belonging to the yeast family, having progressive and infectious features, presenting fungating or warty elevations, with ulceration and the formation of abscesses. Gilchrist was the first to enunciate the view that the ailment was ascribable to vegetable parasites, and has, in conjunction with Stokes, followed up his early researches. Busse, Busche, Sanfelice, Welles, Hyde, Anthony, Herzog, and others have added to our knowledge.

Pathology and etiology.—There is epidermic increase, with projection downwards of pegs and solid masses, penetrating even to the subcutaneous tissue, imparting somewhat the appearance of a squamouscelled carcinoma. In these there occur many small abscesses, in the cavity of which polynuclear leucocytes predominate; and among these are the vegetable parasites, the blastomycetes. These are from 12 to 20 μ in diameter, have a double contour, of which the outer represents the cell membrane, the inner, the retracted cell protoplasm. Some are round, others show budding processes, the usual mode of multiplication. Cultivated on some media, a mycelium may develop, but this has not been observed in the skin. The source has not been discovered. Liebenthal thinks it is always engrafted on a previously diseased soil, but in some cases at least it is primary. While there was a tuberculous history in a few, others sprang from an apparently healthy stock.

Symptoms.—The regions earliest invaded are the back of the hand and the front of the leg, but it may spread widely. The commencement is a reddish, raised spot, which extends in area, and may become pustular. The affected part is more or less covered with warty or papillomatous projections, and as it exudes a sticky, sero-purulent fluid, this dries into investing crusts. The border is frequently reddened, and this may be partially overlapped by masses of vegetations. Sometimes the process ends by the formation of a thin whitish scar, more often it advances with burrowing ulcerations till extensive tracts are involved. There may be no subjective sensations, or burning and pain is complained of.

Diagnosis and prognosis.—On the hand it resembles most closely lupus verrucosus, but no tubercle bacilli have been discovered in sections from pieces excised. The differentiation indeed could scarcely be made from the clinical phenomena. In other situations it reminds of the rare vegetating syphilide, and it is not unlikely that there may be a mixed infection. The prognosis is favourable if attacked early, but the disease has proved fatal in at least one instance.

Treatment.—Iodide of potassium in full doses, and pushed, has proved curative after locally destructive measures had been tried in vain.

ANIMAL PARASITIC DISEASES.

SCABIES.

A DISEASE characterised by itchiness after going to bed, or when heated, presenting multiform lesions, mostly the result of scratching, occasioned by the burrowing of the female acarus scabiei in the horny cuticle. Avenzoar, an Arabian physician, is believed to have known that scabies was due to a mite, and S. Hildegard, in the twelfth century, employed remedies against it; a very clear description of it was given by Bonomo of Leghorn in 1687, yet a general acquaintance with the true nature of the disease only slowly permeated the rank and file of the profession.

Etiology.—The disease is chiefly communicated by sleeping with an infected person, yet as the acarus possesses considerable vitality it is possible that it may live for a time in blankets, etc., and may thus infect a second person. The acarus is just perceptible to the naked eye, and is not unlike though much smaller than the familiar cheese mite. It is roundish and transparent, about one-fiftieth of an inch long, with peculiar triangular, thorn-like projections on the back. The male is considerably less than the female in size. The adult insect has eight legs, all in the male terminating in trumpet-shaped suckers, in the female the posterior one bears a hair. While the species affecting man will live for an indefinite period, if uninterfered with, in his integument—there are cases on record where individuals have never been free from scabies throughout a long life—acari parasitic on other of the mammalia occasionally are temporary tenants, and may cause a good deal of irritation and crusting.

Pathology.—There are only two lesions strictly due to the acarus. One of these is the burrow or tunnel which the female excavates for herself in the middle layer of the horny part of the epidermis, for the male either wanders about on the surface, or at most only digs a superficial cave. The burrow in typical form is a greyish line, little more than the breadth of a hair, straight or winding, and an eighth of an inch or more long. It is often ragged and dirt-stained. The mite herself may sometimes be seen at its blind end as a yellowish grey speck under the cuticle. Behind the mite are eggs and fæces. In very many cases no burrow can be found. The other is a tiny, clear vesicle, situated over the cul-de-sac in which the tunnel ends.

Symptoms.—Itchiness is the first symptom to manifest itself, but it is usually some weeks after contamination, unless several foci have been established at once, ere it becomes aggressive. As the acari multiply, the pruritus spreads over the limbs, anterior part of the trunk, the hands and feet. The sensation begins as soon as the sufferer gets warm in bed, since then the female resumes her progress, and may last all night, effectually preventing sleep. To a slighter extent it occurs in the day if hot.

The situations affected are of importance. The front of the wrists, the forearm and arm, the front of the thighs, the genital organs in men and the breasts in women, the spaces between the fingers, the palms and soles in young people, the nates in children, and the anterior aspect rather than the dorsal of the trunk, are pretty much those in order of frequency. The face is seldom attacked even in severe and long-continued cases. Scratching sets up conditions little different from those of eczema. There may be papules mixed up with wheals. Pustules from secondary infection with pus cocci are not infrequent, more particularly in the careless or neglected. Bullæ, large and yellow, with thick walls, are sometimes seen on the hands or feet, with swelling, œdema, and redness. The lesions are multiform, but preserve a degree of discreteness in arrangement. In cases of long standing very marked pigmentation may arise. At times the symptoms are few and insignificant; this occurs for the most part in those whose habits are cleanly, or who employ soaps containing substances such as carbolic acid, distasteful to the acari. There are rare instances where pruritus is almost wholly absent.

Diagnosis.—This can be established by the discovery of the mite, but in general the fact of itching being principally during the night, the observation of the localities affected, the multiformity and dissemination of the lesions suffice, while it is confirmed if several members of the family are similarly diseased. The complaint which most closely simulates it is dermatitis herpetiformis.

Treatment.—The first preliminary is to administer a warm bath combined with ablution with soft soap. Sulphur ointment, full strength or diluted in the case of children or those with delicate skins, should next be well rubbed in for three successive nights, a second warm bath taken, and the clothes completely changed. If any trace of recurrence takes place, repetition to the itchy part is usually all that is needed. β-naphthol, 1 in 10 of vaseline, lard, or in a vaseline starch paste, can be used in place of sulphur. Or styrax, a couple of drachms, balsam of Peru half a drachm, and simple ointment an ounce, is also efficacious. Care must be taken to avoid over treating.

PEDICULOSIS.

The *pediculus capitis* is so common an epiphyte in the lower orders, that it is regarded as almost a normal tenant. It is oftener seen in children than in adults, and affects chiefly the back and top of the head. Unless numerous, they may produce little more than an annoying itchiness. But if present for some time the scratching induces a moist eczema, the hair becomes matted, sometimes into an inextricable felt-work, a nauseous odour is exhaled, and the cervical and occipital glands enlarge. The best treatment is to saturate the hair with kerosene oil or crude petroleum for thirty-six hours, the head meanwhile being protected by a flannel cap, and caution as to approaching a light being given. The scalp ought then to be well washed with soap and warm water. This effectually destroys the insects and the ova which are attached to the hairs. If this plan is objected to, the unguentum staphisagriæ may be rubbed into the hair.

The integument of certain individuals seems to provide a specially favourable feeding-ground for the *pediculus vestimentorum*. Alcoholic persons, especially if old, are peculiarly prone to these parasites. Many elderly people, too, from carelessness or fear of catching cold, rarely change their under-linen, and somehow acquire them. The insect, while it lives in the clothes, derives its nourishment from the skin. It inserts a long narrow proboscis into a follicle, sucks blood through this haustellum, and on withdrawing it a little wells up into the dilated cavity, giving rise to a minute hæmorrhagic speck, the pathognomonic lesion. Such are found on the back or shoulders. The ova can also be discovered attached to downy hairs. The animals occasion irritation, which induces scratching, and hence excoriations, wheals, pigmentation, and pustulation from the invasion of pus cocci are produced. Treatment consists in thorough change of clothes, washing with carbolic soap, and wearing next the skin day and night a bag containing fragments of sulphur, which at the temperature of the body undergo slow oxidation, and prevent the insects again obtaining a lodgment.

The *pediculus pubis*, though chiefly found about the pubes, may live in the hairy regions elsewhere, as in the axillæ, pectoral hair, and eyebrows and eyelashes. The ova may be seen in wide-spread cases on the lanugo hairs of the trunk. In course of its migrations it produces in some bluish or steel-grey stains, without trace of a puncture. The crab louse causes in most persons much local irritation, and eczematous eruptions. Yet sometimes it produces no pruritus, and no evidence of scratching. They are best got rid of by washing with a storax soap, which dislodges them, and rubbing in stavesacre ointment.

OTHER OCCASIONAL PARASITES.

Various larval forms are occasionally found in or under the skin of man. Thus in cases where an individual is the host of the *Tænia solium*, or from another source, the eggs may be swallowed, and the hydatid developing from them may reach the subcutaneous cellular tissue as its destination. There it gives rise to a rounded or oval tumour, which may attain the size of a pea, is gristly to palpation, and movable. Such may be single or numerous. They are found under the skin of the trunk or limbs, occasion disturbances of sensation, or a feeling of weariness on movement. The diagnosis is difficult, and the presence of the *cysticercus cellulosæ cutis* can only be definitely determined by extirpating one as one would a cyst. Their recognition in the skin may be the means of explaining otherwise obscure symptoms in the brain or internal organs. The grub or larva of various dipterous insects sometimes finds its way into the human skin, and produces red and hard spots, which may become abscesses and burst, discharging the worm; or sometimes thin, slightly raised red lines, which creep onwards, accompanied with severe itching or by pain. A full account of this under the name of *Cutaneous myiasis* has been recently published by Abraham.

Numerous minor pests attack the human integument, and give rise to eruptions and annoyance. Among these may be mentioned the harvest or gooseberry bug, a minute insect which is found in hay fields and gooseberry bushes, and lives for a day or two in the skin. Fleas and bed bugs distress many people exceedingly, scarcely touch others. Mosquitoes fix themselves on new-comers to the regions where they prevail, older residents acquire a degree of immunity. The application of the linimentum saponis to the exposed parts of the body acts as a protective, while alkaline solutions, as aromatic spirits of ammonia, relieve if sopped on to the bitten parts as speedily as possible after exposure. The active ingredient is formic acid, which is neutralised by the alkali.

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LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

SECTION X.

NERVOUS SYSTEM.

INTRODUCTION.

Diseases of the nervous system include maladies of widely different causation and pathological relations. We may distinguish two general classes: those in which there is a primary affection of the nerve structures; and those in which the nerve tissues suffer secondarily in consequence of an affection of the structures which enclose, protect, support, or nourish them—diseases that are chiefly manifested by the derangement of function of the nerve elements that is produced. Between these two classes, however, there are many connecting links. By the second class, diseases of the nervous system are brought into wide pathological relations, and to understand and treat them involves familiarity with many morbid processes, general and local,—with their relations, and with their treatment,—which may be wholly independent of the nervous system itself.

In this class the nerve centres may suffer from disease of the protecting bones, and both the central and peripheral structures from disease of the investing fibrous tissues, the membranes of the brain and cord, and the sheaths of the nerves. All these have pathological relations quite different from those of the nerve tissue. Some of the most important lesions of the nerve centres are dependent on morbid states of the blood vessels, and may be thus secondary effects of disease of the heart, as in embolism—or of disease of the general vascular system, and through this may be the results of the kidney disease, as in hæmorrhage and in softening from atheromatous occlusion. In consequence of other changes in the vessels and membranes, similar lesions are the consequence of syphilis. The various morbid growths are also, for the most part, diseases only in, not of, the nervous system, although one variety of neoplasm affords another illustration of the connection between the two classes; gliomata connect extrinsic growths with diseases that are primarily of the nerve structure. The supporting neuroglia, different from the nerve structures as it seems and indeed is, consists of a residue of the embryonal tissue from which the nervous tissues were developed. In its nutritional tendencies it presents a relation to the nerve structures which, though definite, is opposite; it overgrows when the latter decay, and thus gives rise to the condition of secondary sclerosis. Its growth seems normally to be restrained by the vitality of the higher tissue, and becomes exuberant as soon as that of the latter is lessened. It may persist in tracts, large or small, when there is a congenital arrest of development, such as gives rise to the cavities called syringo-myelia; this persistence is especially important, because the tissue may cease to be quiet, may grow and develop into the definite and dangerous condition known as central gliomatosis. But the neuroglia, as the connective tissue of the central structures, shares the lower morbid tendency of the interstitial tissue of all organs. It is by an overgrowth of this, in random foci, presenting at first some of the features of inflammation, that insular sclerosis develops, while the neuroglia also takes a chief part in all forms of interstitial inflammation in the brain, cord, and nerves. Its secondary overgrowth in degeneration of the nerve structures varies in amount and in energy; even when secondary, it seems occasionally to assume an independence which makes it excessive, and an energy which involves some of the features of inflammation.

We have to distinguish from the primary interstitial changes, those acute processes in which the nerve structures suffer primarily, but with rapidity. When they undergo slow decay, we call it degeneration, but an acute destructive change may take place so rapidly as to resemble parenchymatous inflammation—inflammation in which the primary element is in the proper functional structures of an organ. Such changes belong to the group of diseases that are truly of the nervous system, but are not always readily distinguished from the interstitial inflammations, since the interstitial tissue and vessels may participate in the more intense processes. Their course presents all gradations, from the most acute to chronic forms, but even in the former it is often remarkable how little share the connective tissue elements take. These processes have become, of late, very important, on account of the extent and degree to which they can be traced to toxic influences. They thus constitute, in slow form, the systematic degenerations of the central nervous system, and also, in both slow and rapid forms, the varieties of toxic peripheral neuritis. The former, as well as the latter, have been extensively traced to the influence of blood states. But as chronic degeneration, they are also often due to defective vitality, or imperfect vital endurance, senile or premature, occurring in late life before other structures decay, or as isolated failure soon after development is complete. The latter class is represented by the diseases of which hereditary ataxy is a type.

The toxic influences which are thus effective seem, so far as we can trace their nature, to be chemical. They may be metallic poisons—or the simpler chemical substances of organic origin, as alcohol—or complex organic compounds, either received from without or, more often, formed within the body. They may be likewise formed within the body by the organisms that give rise to acute specific diseases, as has been clearly proved in the case of diphtheria; to the same mechanism many nervous sequelæ of acute diseases are apparently due. These poisons, especially those of organic origin, present the remarkable feature of a special tendency to influence certain nerve structures, which we are only able to distinguish from others by their difference of function; underlying this must be some minute difference of constitution which renders them more susceptible to the particular chemical compounds. These structures suffer most where their vitality is least; for instance, the ends of the nerve fibres,—the parts farthest from the nerve cell of which they are the processes. Thus many toxic agents act on the extremities of the long sensory or motor nerves in the limbs, causing degenerative peripheral neuritis, while a similar degeneration of the fibres of the pyramidal tracts of the spinal cord (the processes of the motor cells of the cortex) constitutes lateral sclerosis. The motor fibres of the limbs are processes of the cells of the anterior grey matter of the cord; the sensory fibres proceed from the cells of the ganglia in the posterior nerve roots. Of the latter, those which seem most prone to suffer are the afferent fibres from the muscles. In locomotor ataxy these nerves suffer first, apparently from a toxic agent which is a result of syphilis, but acts also on the other processes of these ganglion cells—those which become the fibres of the posterior median columns. These ascend the cord, but depend for their vitality on the cells of the posterior ganglia. Thus the degeneration in the spinal cord, the posterior sclerosis, and the changes in the peripheral nerves, are but parts of the toxic influence on one range of cell influence. The afferent nerves from both skin and muscle suffer, however, from many other toxic causes, and, alike in alcoholic neuritis and in diphtheritic palsy, we may have a close resemblance to locomotor ataxy. The susceptibility of the muscle nerves is the cause of the frequency with which the knee-jerk is lost, and of the profound significance of the symptom.

Other toxic agents influence, not the peripheral ends of the long processes of the cells, but the structures in which nerve energy is produced in the central mechanism. Such, for instance, is strychnine, and the strychnine-like products of the organism of tetanus, while one element in the production of the curious disturbance of chorea seems to be the action on the elements of the motor cortex of some poison allied to, but not identical with, that which causes acute rheumatism, and is probably a complex organic chemical compound.

Relative deficiency in the power of resisting toxic agents must be ascribed to imperfect vital energy, a lower degree of the energy of life that maintains nutrition. Such imperfection is manifested by a class of cases in which failure of nutrition occurs as a primary change, either before other parts in late life, as in many cases of spinal muscular atrophy, or labio-glossal paralysis, or senile brain failure, or paralysis agitans,—or else soon after complete development is achieved, as in the so-called hereditary ataxy and its congeners. Moreover, it is possible that a slight difference in the power of vital endurance in some structures causes a greater readiness to suffer degenerative changes in middle life under acquired influences.

The toxic agents which can thus act on the nerve structures and impair their nutrition and function are numerous and varied. They may be derived directly from without, may be produced within the body by the organisms of disease, and may be produced by disordered chemical processes apart from external influence, or by an external influence that becomes effective through a peculiar predisposition. They present mysterious alliances to the agents which act on the fibrous tissues and on other structures. Exposure to cold, apparently the same, may cause in different persons—a general catarrh, local inflammation (as tonsillitis), acute rheumatism (with or without endocarditis), spinal myelitis, or peripheral neuritis, apparently according to the nature of the toxic agent generated, determined by the constitution of the individual; that is, by the whole complex series of reactions that occur when some one derangement disturbs a series in too delicate normal equilibrium. A slight difference in the initial effect may determine a divergence in the chemical processes under the influence of life, and the ultimate result is the same in no two individuals, and may be very different.

To understand the *symptoms* of diseases of the nervous system, the student needs to be specially familiar with certain facts of its anatomy and physiology; the necessary familiarity is not always acquired in the special course of physiology, and generally has to be gained by the application of physiology to disease. It is impossible here to do more than to mention a few of the facts that are of chief importance.

The motor path from the cortex of the brain to the muscles consists of two parts—(*a*) An upper segment (or neuron), consisting of the motor cell of the cortex, its branching processes, and the chief process, which constitutes the pyramidal fibre that passes by the medulla oblongata, through the spinal cord, to the anterior grey substance at a certain level; (*b*) A lower segment, the motor cell of the anterior horn, its branching processes that come into relation with the endings of the pyramidal fibre, and its chief process, the nerve fibre which passes to the muscle and there divides and ends on the muscular fibres. Disease of either segment causes loss of voluntary power. The lower segment is also part of the reflex arc, and on it, moreover, muscular nutrition depends. Hence disease of this segment causes muscular wasting, accompanied, if rapid, by the reaction of degeneration to electricity, that is, the loss of the faradic excitability due to the nerve fibres, and persistence of the voltaic excitability due to the muscular tissue itself. Such disease also arrests reflex action, alike that which is due to impulses from the muscles, as the knee-jerk, and that from the skin. Disease of the upper segment, while equally lessening motor power, does not interfere with muscular nutrition or reflex action, and, indeed, permits the latter to become excessive, so that the normal knee-jerk is increased and an abnormal clonus can be obtained, and the normal reflex tone is increased to tonic spasm.

The sensory path is by the ganglia of the posterior roots, and on the cells of these ganglia the afferent nerve fibres depend for their vitality. The minute differentiation of cutaneous sensibility involves, at first sight, a puzzling multiplicity of distinct and separate function, but it becomes intelligible when we perceive that each of the axis-cylinders of the nerve fibres is a bundle of separate conducting fibrils, a fact which will be again referred to. The impulses conveyed by the afferent fibres from the muscles seem to pass from the cells of the posterior ganglia, some to the grey matter of the cord (of which some subserve the muscle-reflex action), others by the fibres which constitute the posterior median columns to the medulla oblongata, and their impulses pass, in part at least, to the co-ordinating middle lobe of the cerebellum. Hence disease of all these afferent root fibres from the muscles arrests both the spinal and cerebellar co-ordination, causing ataxy, with loss of the knee-jerk. But disease of the posterior median columns in the dorsal region causes unsteadiness, closely resembling that of cerebellar disease, by interrupting the impulses from the muscles to the cerebellum, but it does not arrest the spinal reflex process and the knee-jerk. The co-ordinating function of the cerebellum is subserved only by the middle lobe, and the known facts suggest that here the impulses from the muscles are combined with those from the semicircular canals of the ear, and with other impulses, due to the action of the centres which control the position of the head and eyes, and afford guidance of the relation to the body of seen objects. The facts also indicate that the effect of these combined impressions on muscular co-ordination is due to an upward influence, from the cerebellum, on the motor cortex of the cerebral hemisphere, in which the nerve impulses for the muscles are primarily arranged.

The sense of posture appears to be the effect on consciousness of the activity of those motor centres; and vertigo, at least in its typical form, is due to their derangement; involuntary turning (actual vertigo) is due to a greater degree of the process that causes a mere sense of turning (subjective vertigo). Although the impulses from the muscles influence consciousness when they are excessive, and cause very definite sensations of pain, they do not seem, in normal degree, to produce any direct sensation. The sense of posture seems chiefly the result of the effect on consciousness of the degree of activity in the various motor centres regulated by the afferent impulse. It is thus the effect of a motor process. If this is perceived, many anomalous facts can be understood.

The region of the cortex from which the motor impulses pass down to the spinal cord is the only part of the cerebral convolutions in which destruction of tissue causes lasting *loss of power*. This loss, however, is persistent only in those movements that are not bilateral. In proportion as movements are bilateral they are represented in both hemispheres, and can be innervated from either. But it is probable that these motor processes are excited by others, which we cannot discern, in other parts and structures of the cortex.

We have also reason to suspect that only permanent *loss of sensation* is due to disease of the parts of the cortex to which the impulses first arrive (although some impairment is produced by disease of the motor cortex). We have, indeed, evidence of this only as regards the visual impulses in the half-vision centre in the occipital lobe, but it is probable that the arrangement is similar for all sensations. It is important to note such indications, even when they are only suggestive, because the area of the cortex is very large regarding which we have no definite knowledge of function. In the region in which loss of structure causes no definite limited loss of function (as it does in the motor region and in the occipital half-vision centre), the effect of loss of structure seems to be to lower the function as a whole. This is a mysterious fact which we can only note and remember. In these parts, moreover, irritation may cause discharges that act on consciousness, and these may teach us something of their function, although in language which is difficult to interpret; we meet with these in the aura of epilepsy and the premonition of migraine.

One other subject of great importance must, however, be mentioned. Recent researches compel a fundamental change in our conceptions of the structure and function of the central nervous system—a change of the utmost importance to pathology. A nerve cell—such, for instance, as one of the motor cells of the cord—has many processes, of which one is larger than the rest, and becomes clothed with white substance, as a nerve fibre, while the others quickly branch, and their ramifications are lost in the spongy grey substance. In this they have been hitherto believed to be continuous with similar ramifications of other nerve cells, or of the extremity of a long nerve fibre. But it has been found[1] that these branching processes are not continuous with others, but end, their endings being contiguous to, but not continuous with, those of other processes or fibres. This involves a complete difference in our conception of their action. They do not merely conduct; the impulses which arise in them must be excited afresh, and not merely transmitted. They must be excited by the activity of the contiguous terminal ends of branches of other cells or of fibres, as are the nerve fibres, for instance, in the skin by external energy. This involves the conception that the nervous system is made up of units, each dependent on a nerve cell, and consisting (1) of the nerve cell; (2) its branching processes, which have been called dendrons or dendrites; and (3) its chief afferent process, which has been called the neuraxon or axon. Following Waldeyer, the term neuron, with a plural according to the language of the country (in English neurons, in Germany neuronen),[1] has been applied to each nerve unit.

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[1] By the researches of Waldeyer and others, of which an important summary and extension, based on original work, was given by Ramon y Cajal in the Croonian Lecture to the Royal Society, 1894.

The importance of this discovery is increased by the fact that the axis-cylinder of the chief fibre seems to be composed of fibrils, which separate in the cell, and pass to different dendrons, and that these branching processes present a similar fibril structure. Apparently most of their fibrils pass into the cell towards the axon, but some turn back to other dendrons. There is considerable evidence that the fibrils pass through the cell without interruption. Hence we must give up the attractive doctrine that the nerve cells generate nerve force. Their function must be conceived as nutritional only, apparently by means of the continuity of their protoplasm with that around the processes and fibres which is reinforced at intervals by nuclei and the protoplasm around them.

Similar neurons in the cortex of the brain probably subserve the connection between its several parts, and we must assume that others, whose axons are the fibres of the corpus collosum, connect like regions in the two hemispheres. Indeed, the same plan of minute structure must be assumed to be universal in the central nervous system.

For the origin of nerve impulses we must therefore look to the finely divided nerve substance at the extremity of the dendrons, in the spongy grey matter, the numerous branchings of which make the total quantity of this terminal nerve substance very large. Moreover, the fine division brings it into perfect relation to the nutritional plasma, from which the molecules lost in function are at once renewed. Difficult as it may be at first to conceive these endings as the seat of the change of latent chemical energy into the peculiar and still unknown form which is propagated as a nerve impulse, it becomes less difficult when we note that at the periphery similar nerve impulses arise at the extremities of the branches of the nerve fibres in the skin. In these the impulse is excited by a stimulus, and so, as just stated, we must conceive the impulses to arise in the dendrons. It is excited in their terminations by the nerve impulse in the contiguous terminations of the branches of other cells. It may at first seem difficult to conceive the existence of precise paths between the fibrils which are only in terminal contiguity, but this difficulty is lessened when we remember that the stimulation of muscular fibres is effected with absolute precision, and yet there is absolute discontinuity of structure between the terminations of the nerves and the muscular protoplasm. The difficulties of the new doctrine are unquestionably less than the difficulties which it displaces. The importance of these conceptions in relation to the pathology of disease will be sufficiently evident when they are considered in detailed application.

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[1] The use of the term neuron as a designation for the whole of the nerve unit has become so well established in all countries, that it is impracticable now to restrict it, in accordance with etymology, to the process which becomes a nerve fibre, and the attempt would cause useless derangement of terms and much confusion of thought.

The *diagnosis* of diseases of the nervous system needs a clear conception of their causes and of the mode in which the symptoms are produced. An illustration of the need for knowledge of the morbid influences is presented by the facts respecting toxic agents, which have been already referred to. Their action on the nerve structures is always symmetrical. Not only is it symmetrical, but, except when intense, it is limited in incidence to structures having corresponding functions. This combination of functional limitation and bilateral symmetry is of extreme diagnostic importance. Its meaning is that the cause acts through the blood, because it reaches the similar structures on both sides.

This fact presents an illustration of an important principle of diagnosis applicable to almost all forms of disease. Unless a case obviously conforms to some familiar type, it is necessary, in order to obtain a secure diagnosis, to discard types and names, and to reason out the case as if it were an entirely new problem. The symptoms are evidence of the seat of the morbid process; that is, the derangement of function is evidence of the structures that are damaged. The mode of onset of the symptoms is the chief indication of the nature of the morbid process which they indicate; and, lastly, the presence of a known cause of such a morbid process gives additional probability to its activity. This diagnostic method needs only a secure knowledge of a limited number of facts, physiological and pathological, but there must be implicit trust in that knowledge, and in the process of reasoning. Then an accurate opinion can be formed in at least four-fifths of the cases of disease which now baffle many practitioners. In many forms of degenerative and organic disease, as well as in the various nutritional and functional affections, the types that are described, by which alone the diseases can be learned in the first instance, represent simply the more common groups of systems that are produced, and these are also met with in combinations and character that present every degree of variation. Hence a large number of the cases correspond to no described type, and could not be included in definite types, however numerous these were made. By reasoning out the case in the manner described, the diagnosis becomes sure and independent, and what is still more important, those elements of the case are directly perceived which alone can afford guidance in treatment. A moderate amount of deliberate practice of this diagnostic method will speedily enable it to be employed without difficulty. When the diagnosis of seat and nature has been reached, the correspondence with type and name may then be considered, but it must not be forgotten that those who are most familiar with diseases of the nervous system meet daily with cases which conform to no type, the precise like of which they have not seen before, and the nature of which they can only discern by the method described. That which is true of some diseases to the experienced physician is true of a larger number to the practitioner, but the end to be attained is the same, and so is the road to it.

For the *prognosis* and *treatment* of disease, it is essential above all to form a mental picture of the malady, and to endeavour to see, with the mind's eye, how the symptoms are produced by the primary morbid process. Thus alone can a forecast of the future be made, and its limitations seen; thus alone can the range of treatment be discerned, and its means be wisely ordered. For instance, a man two years after syphilis is seized with sudden hemiplegia. The morbid process must be a vascular lesion, on account of the sudden onset, and the age of the patient suggests the occlusion of an artery and softening, rather than rupture and hæmorrhage. With this the symptoms of the onset agree. From the absence of any cause of embolism, and from the presence of syphilis (the two common causes of arterial occlusion at this time of life), the conception formed is that syphilitic disease, thickening the wall of an artery, has permitted the occurrence of sudden thrombosis in the trunk or in a branch, the orifice of which is narrowed. The mental picture that is formed is that of complete occlusion of the trunk or of the branch, with necrotic softening of the area of the brain supplied. This picture reveals to the mind at once the degree to which the prognosis is influenced by the fact of the syphilis. The syphilitic disease is in the wall of the artery. The vessel is closed by clot, which has no relation to the syphilis, while the symptoms depend on the necrosis due to the clot, which does not differ from that which results from occlusion due to any other cause. The picture thus formed will at once reveal the fact that no treatment of the primary disease of the arterial wall can influence the symptoms that have resulted from its ultimate consequence. The error will be avoided of ascribing any slow improvement which would occur equally in a case of embolism, to the iodide of potassium that may be given, but it will also suggest the probability that the artery occluded is not the only one that is affected, and the great importance of this treatment. Again, a patient presents evidence of a tuberculous tumour in the motor region of the cortex; under tonic treatment, commencing optic neuritis subsides, headache ceases, and it is evident that the tumour has become quiescent and is shrinking. But it has caused repeated convulsions, so severe as to be general, and these persist. The mental picture should present the motor structures in the cortex as so damaged by the growth, that although they have partly recovered, they have remained so damaged as to be unstable, liable to discharge, an instability kept up by the influence of the repeated discharges. Hence it cannot be expected that the diminution of any hemiplegia that has been caused by the growth will be accompanied by corresponding cessation of the fits. Again, a patient has an attack of facial paralysis from cold, that is, from neuritis within the bony canal. A fortnight later it is found that taste is unaffected, and the mental picture should be that of an inflammation that does not extend up to the place at which the chorda tympani leaves the facial nerve. Moreover, electrical irritability is found unchanged, and this should cause the nerve fibres to be thought of as undegenerated, and therefore only slightly compressed by the swelling of the inflamed sheath. By that time the active stage of the inflammation is over, the swelling will be subsiding, and an early return of power may be confidently anticipated. We conceive such a condition, indeed, without being aware of a mental picture, but this is often unconsciously formed, and a conscious effort to produce it will add greatly to the practical power of discerning alike the future course and the room for present treatment. The same habit should be customary in the cases of many other conditions, such as myelitis, peripheral neuritis, etc. It is thus that the method of diagnosis that has been urged is especially serviceable, because it enables the lesion to be conceived, and mentally portrayed, far more effectively than if types and names are the first steps. Especially important is it to recognise on what the symptoms immediately depend. In almost all the diseases that are only in, and not of, the nerve structures, the symptoms depend upon simple changes in the latter, the same whatever be the nature of their cause. The changes in the spinal cord which cause paraplegia may be identical, although their cause is a cancerous growth, a syphilitic tumour, or an aortic aneurysm.

The *treatment* of the two classes of disease, mentioned at the outset, differs in its scope, its degree, and its character. The diseases that are only within the nervous system damage structures that are, for the most part, free from morbid tendency, and it is doubtful whether their recovery can be appreciably influenced by treatment beyond the simple measures called for by secondary inflammation wherever it is seated. The important element is the treatment of the primary disease, to lessen it if possible, or to prevent its increase and that of its effects. A minute difference in the degree attained by the primary process before arrest may involve a vast difference in its effects and their duration.

The diseases that are actually of the nerve elements present some scope for direct treatment, although this varies widely in different cases, and is too often small. It is certain, however, that the greater the perseverance, the less the tendency to give up the task—the greater will be the frequency with which definite good is achieved.

The remark just made regarding the effect of perseverance applies to all therapeutic measures designed to influence the nerve structures, and, among others, to the use of chemical agents. Their choice must depend for the most part on experience, on the knowledge derived from trial and observation. The influence of chemical agents on these diseases, long recognised, is intelligible when we consider the vast influence such agents have in the causation of these diseases. If some chemical agents can derange, other agents may restore. Experience confirms the expectation that this field of therapeutics will yield results wider and more important than is sometimes now thought.

As in all diseases, emphatically in those of the nervous system, no element of treatment can be disregarded that may have an influence for good. Moreover, treatment that is powerless alone may be useful in combination, and that which does no good at one stage may do good at another, and, of two cases apparently similar, one may be susceptible to treatment that is ineffective in the other. It should be needless to insist on the importance of that which may be called the common sense of medicine—wise diet, regulated exercise, fresh air, and all those influences that promote general systemic health. Counter-irritation is often useful, and, in various ways, massage and electricity are of service. But massage is often overdone; it is often conceived to have far greater influence than it possesses, and, in its modern development, it is elaborated to a degree that is not altogether devoid of harm. There is too much neglect of the upward movement of the rubber's hand, which has so great an influence on the movement both of the blood in the vessels and of the fluids in the lymphatic spaces. The over-development of massage has had, however, the effect of reducing electricity to its proper place in professional esteem, which is chiefly the maintenance of functional capacity in muscles, while the nerve influence is, for a time, in abeyance, and the relief of pain in some, not numerous, cases.

There are two preventive measures which need to be specially insisted upon. One is the danger that muscular contractions may develop insidiously in the opponents of paralysed muscles. If the danger is known, and watch is kept, they may be prevented with a tenth of the difficulty needed to get rid of them. The second is the danger of the unperceived development of vesical incapacity. In all forms of spinal cord disease, and in some cerebral diseases, it is common, through imperfect contraction of the bladder, for residual urine to occur and to increase more and more, unconsciously to the patient, and to induce organic changes in the kidneys, probably by a hindrance to the flow of urine from the ureters and habitual increased pressure within the organs. This is often the cause of mysterious pyrexia, and too often of sudden toxic symptoms and speedy death—effects that can be surely prevented by the early recognition of the state, and the careful use of the catheter, with scrupulous antiseptic precautions.

Lastly, it cannot be too strongly urged that the chief opportunity for the effective treatment of all acute diseases of the nervous system rests with the practitioner, who alone sees them in the early stage, and alone can bring treatment promptly to bear upon them. This is conspicuously true, for instance, of all sudden cerebral lesions; in these the immediate adoption of means to lessen the tendency to continued hæmorrhage from an artery, or to the extension of clot within it, may determine whether the patient shall live or die, whether the effect shall be transient or lasting palsy. The same fact is not less conspicuous in the acute inflammations of the spinal cord, in which the simple treatment of the morbid process on the principles on which any local inflammation should be treated, may determine a vast difference in the degree of paralysis that results, and even whether it shall be transient or permanent. A very slight difference in the degree of damage to the conducting fibres may involve recovery or destruction. So likewise it is certain that the prompt recognition and energetic treatment of the indications of syphilitic vascular treatment, *e.g.* of the symptoms of lessened circulation through one of the cerebral arteries, may prevent its occlusion and the enduring consequences.

In the chronic maladies, treatment frequently fails to counteract the progressive tendency of the morbid process, which is often strong. And yet, even in these, much more is often achieved than is commonly thought, and retarded progress, or even arrest, may be a result far more satisfactory than it seems to the sufferer. In the nutritional and so-called functional diseases, however, the effect of treatment is often such as to make life definitely enjoyable, instead of barely endurable, and the gratitude obtained is no small encouragement to persevering effort, even in cases that seem at first most obstinate.

Finally, it is well to remember, and often wise to impress, the fact that, next to knowing what can be done, it is important to know what cannot be done, and so to save waste of time, of money, often of strength—all of which may be more wisely used. It is often well also to enforce the fact that measures which cannot do good can often do grave harm. And it is well for the practitioner to remember that those diseases which are not only in but of the nervous system are among the maladies whose course is most uncertain, and that the cases are rare in which it is justifiable to absolutely exclude hope at least of life, and that there are perhaps no cases before which it is right to sit with folded arms.

W. R. GOWERS.

DISEASES OF THE NERVES.

THE OLFACTORY NERVE.

The olfactory nerve may be paralysed as the result of injury from falls or blows on the forehead or occiput; from disease of the frontal or ethmoid bones; from meningitis of the under surface of the frontal lobe; from pressure due to hydrocephalus or tumour, or direct involvement in the latter; from neuritis; from degeneration due to old age or locomotor ataxia. Anosmia is rarely caused by a lesion in the cortical centre for smell (the uncinate gyrus, according to Ferrier). It may be due to congenital defects, or may form part of a hysterical hemianæsthesia. It may also result from excessive stimulation of the nerve by very strong odours.

Anosmia, or loss of the sense of smell, may be due either to lesions of the olfactory nerve, its peripheral terminations or cortical centre, or to affections of the mucous membrane of the nose. Anosmia is most frequently caused by chronic catarrh of the nose, atrophy of the mucous membrane, ozæna, polypoid growths, excess of secretion, and by changes in the mucous membrane secondary to disease of the fifth nerve; these causes, however, do not come under consideration here. Hyperosmia, or excessive development of the sense of smell, sometimes reaches an extraordinary degree. It is generally a manifestation of hysteria, or of a highly neurotic temperament. Parosmia, or perversion of the sense of smell, is most frequently an insane hallucination, or it may form the aura of an epileptic fit. It occasionally results from injury to the head. In testing the sense of smell, pungent substances, such as ammonia, which stimulate the fifth nerve, should be avoided. Oil of cloves, peppermint, asafœtida, brandy, or whisky may be used.

The prognosis is, as a rule, unfavourable, and the treatment unsatisfactory. If the anosmia is due to a nerve lesion, an attempt may be made to restore the function by the use of iodide of potassium in suitable cases, or the insufflation or injection of strychnine in exceedingly minute doses. The local application of even weak galvanic or faradic currents is seldom well borne.

THE OPTIC NERVE.

The retina and the macula lutea may each be divided into four quadrants by a vertical and a horizontal line passing through the centre of the macula. In the optic nerve the macular fibres occupy a central position, being surrounded by the fibres of the four quadrants of the retina, these fibres having almost the same relative position as in the eye. At the chiasma there is a semi-decussation of the nerve, in such a way that the fibres from the outer half of each retina (and macula) occupy a lateral position and pass into the optic tract of the same side, while those from the inner halves of the retina (and macula) cross into the opposite tract (Fig. 1). In this way each optic tract obtains fibres from the corresponding halves of both retinæ, the fibres from the right halves of the two retinæ passing into the right optic tract, and from the left halves into the left optic tract.

The optic tract terminates, by three distinct bundles, in the anterior corpus quadrigeminum, the external corpus geniculatum, and the pulvinar of the optic thalamus respectively. From these three ganglia fibres pass by way of the optic radiation of Gratiolet to the occipital lobe. Those from the geniculate body appear to form the visual path, and, according to Henschen, terminate in the sides of the calcarine fissure. The cortex in this region is therefore in relation to the corresponding halves of both retinæ and to the opposite halves of the visual field. The bundle which passes to the anterior corpus quadrigeminum forms the path for the reflex action of the pupil under the stimulus of light. The function of that part of the tract which enters the pulvinar is not yet definitely understood. Lesions limited to this part of the optic thalamus do not produce any disturbance of vision or of pupillary reaction.

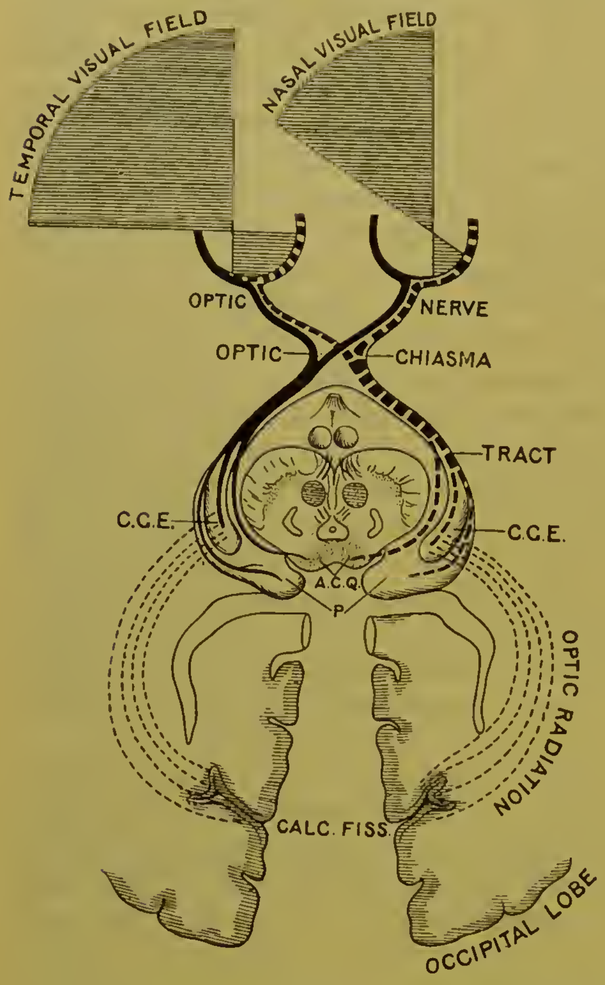


Fig. 1.—Scheme showing how each half of the field of vision is related to the opposite occipital lobe and its calcarine fissure (CALC. FISS.); also the primary terminations of the optic tract in the anterior corpus quadrigeminum (A.C.Q.), the external geniculate body (C.G.E.), and the pulvinar (P.).

Optic Lesions.

Etiology and pathology. — The most important lesions which affect the optic nerve within the orbit or within the cranial cavity are:—Neoplasms, cellulitis or abscess, interstitial or parenchymatous neuritis, meningitis, and aneurysm of the ophthalmic or internal carotid artery.

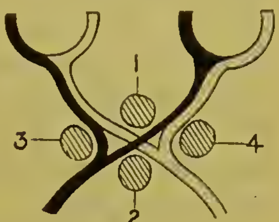
The chiasma is most commonly affected by tumours (especially those of the pituitary body, as in acromegaly), by pressure of the distended infundibulum of the third ventricle in internal hydrocephalus, by tuberculous and syphilitic growths, and by chronic inflammation of the dura mater. The optic tract may be involved in tumours of the temporo-sphenoidal lobe or basal ganglia, or in disseminated sclerosis. The intra-cerebral visual path may be interrupted by softening, hæmorrhage, or tumour in the basal ganglia, the occipital lobe, or even the hinder part of the parietal lobe (the angular gyrus, for instance). Henschen has shown that, in order to interrupt the visual path, a cerebral lesion must destroy either the lower fourth of the optic radiation of Gratiolet or the grey matter forming the calcarine fissure. A lesion in the angular gyrus will not cause hemianopia unless it extends deeply enough to involve the optic radiation.

Symptoms.—**Optic nerve.**—Complete destruction of one optic nerve will cause complete blindness in the corresponding eye, with loss of the pupil reflex when light is thrown upon the blind eye.

Affection of the central fibres of the nerve produces a defect in the centre of the field of vision (central scotoma), which may be either more or less complete, or involve merely the perception of red and green. This condition is found in some forms of toxic neuritis, such as that produced by diabetes, by the excessive use of tobacco or alcohol, or both combined, and by some of the acute infective diseases. This retro-bulbar neuritis may be acute or chronic, is generally bilateral, and occurs after the age of thirty-five. If the periphery of the nerve is affected, as by a neuritis spreading inwards, then the field of vision is contracted concentrically. If the nerve is irregularly affected, then the field of vision will be irregularly reduced. The pupil reflex will vary according to the power of the nerve to convey impulses to the oculo-motor centre. To the ophthalmoscope the disc may appear normal or may show evidence of simple atrophy.

The lesion which affects the optic nerve may also involve the motor nerves of the eye and paralyse some or all of them, or it may involve the branches of the fifth nerve and cause neuralgia or anæsthesia.

Optic chiasma.—The blindness produced by disease of the chiasma varies with the situation and extent of the lesion. If this is situated either behind or in front of the chiasma (1 and 2, Fig. 2), the decussating fibres of the nerve, and therefore also the nasal halves of the maculæ and retinæ, will be involved. There will be blindness, therefore, in the outer half of the field of vision in either eye (temporal hemianopia). A lesion on the outer side of the chiasma (3 and 4, Fig. 2) will affect the uncrossed fibres of the nerve, will paralyse the outer half of the retina of the same side, and produce blindness on the nasal half of the visual field of that eye. A symmetrical lesion on both sides of the chiasma (3 and 4, Fig. 2), as in Knapp's case of calcification of both internal carotids, will cause nasal hemianopia. A lesion involving the decussating fibres in the chiasma and the uncrossed fibres on one side will produce complete blindness in the whole field of the retina on one side, and in the temporal half of the field in the other (Fig. 3). If the whole chiasma is destroyed, the blindness will be complete in both eyes (Fig. 4).



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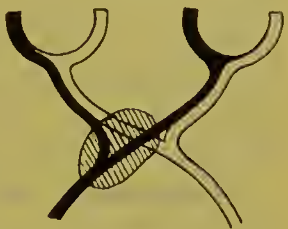

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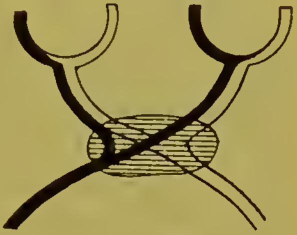

4.

Figs. 2, 3, and 4.—To show how lesions affect the optic nerve, tract, and chiasma, and consequently the field of vision.

Ophthalmoscopically the disc may be normal in the early stages, or it may show signs of optic neuritis, especially if a cerebral tumour be present. Slight atrophy may appear after a longer or shorter time.

Optic tract and visual tract in the occipital lobe.—A lesion of either of these will interrupt the fibres from the outer half of the retina of the same side and inner half of the retina of the opposite side; and will therefore produce blindness in the halves of the field of vision opposite the seat of the lesion. If, for example, the right optic tract is affected, then the right halves of the retinæ will be blind, and vision will be lost on the nasal half of the right field and on the temporal half of the left (Fig. 1). This condition is called homonymous hemianopia. The area of field affected varies in different cases. Most commonly it extends quite to the middle line, but as a rule the margin diverges round the macula so as to include it in the seeing half. Variations from this are common, and depend, in cases of complete hemianopia, on individual differences in the distribution of the fibres in the retina (Fig. 1). The patient may be unaware of his defect. He may unconsciously keep his head turned a little towards the blind side so as to fix objects with the portion of field remaining.

The ophthalmoscopic appearances vary in different cases. The disc may be normal, or it may show changes depending on the nature of the lesion producing the hemianopia. Thus a cerebral tumour may produce optic neuritis, or hæmorrhage during chronic Bright's disease may be associated with albuminuric retinitis. Associated symptoms may also be present, and vary according to the situation of the lesion. When this affects the optic tract as it passes round the cerebral peduncles (Fig. 1), it may produce hemiplegia on the opposite side of the body, *i.e.* on the side on which the patient is blind. More rarely, there may be hemianæsthesia, and in cases of right-sided hemianopia there may be peculiar defects of speech.

It may, in rare cases, be possible to diagnose the seat of the lesion by means of Wernicke's *hemiopic pupil reaction,* a test which it is very difficult to employ satisfactorily. When the tract is involved, light carefully thrown on the blind half of either retina will not produce a contraction of the pupil, because the fibres to the corpora quadrigemina are interrupted. If the lesion be in the corpus geniculatum, and do not affect the fibres to the corpora quadrigemina, the pupils will contract.

Diagnosis and prognosis.—The seat of the lesion is determined by the symptoms above described. They may be briefly recapitulated as follows:—

Disease of the nerve will produce either total unilateral blindness, central scotoma, or peripheral or irregular contraction of the field of vision. Disease of the chiasma generally produces temporal hemianopia, blindness in both eyes, or complete in one eye and affecting the temporal half of the field of vision in the other. Nasal hemianopia—a rare condition—is also due to affections of the chiasma. Disease of the tract always produces homonymous hemianopia, which may be differentiated from the hemianopia due to disease of the cerebrum by associated symptoms, such as hemiplegia and Wernicke's hemiopic pupil reaction. The prognosis is usually unfavourable, the blindness seldom passing away entirely, except in the toxic and rheumatic affections of the nerve, or where its paralysis has been due to removable pressure.

Treatment.—This must be in each case directed towards the removal of the causal condition. In tobacco amblyopia, complete abstinence from tobacco and alcohol is necessary. Iodide of potassium is sometimes of use, and strychnine, injected subcutaneously, is of value if the cases have not progressed to atrophy. When the atrophic stage is reached, treatment is of little avail.

OPTIC NEURITIS.

Etiology.—The most common cause of optic neuritis is intracranial tumour, which produces a higher degree of neuritis than any other condition, and is accompanied by it in more than 80 per cent. of the cases. Meningitis (tuberculous, simple, or purulent) is the next most common cause. Neuritis is also present in some cases of cerebral abscess, cerebral or meningeal hæmorrhage, and chronic cerebritis, even when no naked-eye change is present. It may also be produced by chronic hydrocephalus, aneurysm, or embolism (especially if septic) of the internal carotid artery and thrombosis of the cavernous sinus. It is rarely found in tumours of the medulla or in simple meningitis of the vertex. It has been seen, though seldom, in myelitis, disseminated sclerosis, general paralysis, and chronic insanity. Neuritis may be due to local disease within the orbit, and it occasionally occurs in certain toxic and infective diseases, such as syphilis, Bright's disease, the acute specific fevers, malaria, chlorosis, and menstrual disorders.

Pathology.—There is no space to indicate here the various views entertained as to the pathology. It is now known, however, not to be dependent on mere mechanical pressure. Most probably it is the result of the action of toxic substances formed by tumours or other products on the vessels of the nerve, leading to their dilatation, the emigration of leucocytes from them, and to the degeneration of the fibres.

Symptoms.—The external appearance of the eye is normal; and it is only on ophthalmoscopic examination that any morbid change is seen. In early stages the optic disc or papilla is much redder than usual, sometimes greyish red or bright red, and almost indistinguishable from the choroid. Its margins are blurred and indistinct or "woolly," and the whole disc is considerably raised and swollen, so that the normal physiological cup is quite obliterated. When examined by the direct method, the blurring of the disc is seen to be due to a fine radiating striation. This is due to congestion of the vessels, and sometimes, later, to degeneration of the nerve fibres and inflammatory exudation around them. The emerging vessels are curved over the swollen papilla, so that the parts which dip down towards its periphery are not well seen. The veins are greatly dilated and tortuous, and in later stages the arteries become very narrow, and appear at parts to be lost in the infiltration in the disc. Hæmorrhage may appear in or near the papilla, dark in colour and flame-shaped, or large and elongated.

These appearances are usually present in both eyes, may develop rapidly, in a fortnight or less, or may take months to reach their acme. The severity of the condition is proportionate, as a rule, to the rate of development. When the acute stage is past, there is either complete recovery, the vascularity disappearing, and the veins and disc again becoming normal, or, in severe cases, the neuritis may be succeeded by atrophy (consecutive atrophy). In this condition the new tissue formed in the acute stages cicatrises and compresses the nerve fibres, thus rendering the disc white and atrophied. There may also be areas of degeneration in the neighbouring parts of the retina, and sometimes streaks of white along the vessels. The degree in which vision is affected varies greatly. In some cases it remains perfect until a late stage, but it may be lost suddenly and early, especially in cerebellar disease. The field of vision may remain normal for some time, but sooner or later becomes contracted. The contraction may be concentric and regular, or hemianopia may be present. The colour-sense is often defective, red and green being the first colours lost. The reactions of the pupil are maintained as in health, unless the neuritis causes complete blindness, in which case the iris light-reflex is lost.

Diagnosis and prognosis.—The diagnosis of optic neuritis can only be definitely determined by ophthalmoscopic examination. In the early stages, when the changes in the disc are slight, it may be difficult or impossible to decide, but in late stages, when the appearances are well marked, the condition is unmistakable. It should not be forgotten that hypermetropia may cause congestion or even slight neuritis. The prognosis depends upon the cause of the condition. It is favourable when the neuritis is due to a remediable lesion, such as a general disease, especially syphilis, or to a cerebral tumour or abscess which is amenable to surgical treatment. It is very grave, or even hopeless, when due to tumours which cannot be removed, the condition in these cases invariably progressing steadily to a fatal termination.

Treatment.—The treatment of optic neuritis varies with its cause. Surgical interference, when possible or advisable, is the only treatment for tumour or abscess of the brain, while meningitis is in most cases beyond remedy. Mercury and potassium iodide should be given in syphilitic cases, iron in chlorosis, salicylic acid in rheumatism. It has been found recently by Horsley and others that optic neuritis has been arrested or improved by simple trephining, even in cases of cerebral tumour which could not be removed by operation.

CHRONIC RETROBULBAR NEURITIS.

Etiology and pathology.—This condition is most frequently due to the excessive use of tobacco, especially when combined with the abuse of alcohol. It may be also due to diabetes, gout, influenza, or syphilis, and to the action of lead, bisulphide of carbon, quinine, and some other drugs. It arises occasionally in tabes and disseminated sclerosis, possibly as a result of the combined action of the disease and excess in alcohol or tobacco.

Both eyes are usually affected. There may be merely a diminished acuity of vision, or a characteristic central, oval scotoma, which may vary in degree from a slight loss of colour-sense to a complete absence of vision. The periphery of the field remains normal as a rule. Ophthalmoscopically, the fundus may be normal in the early stages; later, it may be hyperæmic and indistinct at its edges, then the disc may be greyish white in the temporal half, and ultimately become entirely atrophied. The pathology of the process is for the most part a parenchymatous neuritis—more rarely it is interstitial.

The diagnosis is made from the presence of the central scotoma. The prognosis is good if the cause can be removed, and if atrophy is not too far advanced. Recovery of complete vision may not be attained for weeks or months.

Treatment.—The treatment should be directed towards removal of the cause, improving the general health by tonics, and improving the nutrition of the nerve by iodide of potassium in suitable cases, and by hypodermic injections ($\frac{1}{60}$ - $\frac{1}{40}$ gr.) of strychnine.

OPTIC ATROPHY.

Etiology.—According to its various causes, atrophy of the optic nerve has been classified as primary, secondary, and consecutive.

Primary atrophy.—Nearly one half of the cases of primary atrophy appears in some spinal lesions, namely, in locomotor ataxia, insular sclerosis, Friedreich's ataxia, and after injury to the cord. It is also found in general paralysis, and in certain toxic or infective diseases, such as syphilis, malaria, diabetes, poisoning by narcotics, quinine, and iodoform. It has also been attributed to exposure to cold, to malnutrition, or to menstrual disorders. Locomotor ataxia is undoubtedly the commonest cause of the primary form of optic atrophy. The atrophy is often a very early symptom of the disease, and may precede the spinal symptoms by a long interval, sometimes as much as ten years. There may be an early grey degeneration with full acuity of central vision and slight contraction of the field for form and colour.

Secondary atrophy is due to some direct pressure on the optic nerve, chiasma, or tract, by tumour, inflammatory exudation, distension of the third ventricle, or by a splinter of bone after a fracture of the base.

Consecutive atrophy is produced by the cicatricial contraction of the new tissue thrown out in acute papillitis.

Embolism of the central artery of the retina and certain diseases of the retina may also produce atrophy of the optic nerve.

Pathology.—The pathology of the disease differs according to the cause. In primary atrophy there is a parenchymatous degeneration of the nerve fibres. They lose their medullary sheath, become shrunken and granular, and show fine fatty particles, compound granular corpuscles, and amyloid bodies. In secondary atrophy there is an interstitial neuritis, the septa being increased in number and size, and the nerve fibres diminished. The nerve may finally be converted into a fibrous cord. In consecutive atrophy there is a more active interstitial neuritis, with a great increase in connective tissue nuclei and young connective tissue, which, in later stages, contracts and compresses the nerve fibres.

Symptoms.—The appearance of the disc varies somewhat in the different types of optic atrophy. In primary atrophy the disc is sharply outlined, in colour papery white-grey, or grey-red, often becoming cupped. The lamina cribrosa is visible as distinct mottling. The vessels are uncontracted in the early stages, and are free from white lines. In secondary atrophy the appearances are very much the same, but the vessels, especially the veins, are contracted. In consecutive atrophy the disc is hazy in its outline, and very white, the lamina cribrosa being quite lost. The veins are large, tortuous, and bordered by white lines, while the arteries are small and contracted.

The changes in vision vary greatly in different cases. Central vision may be only slightly impaired, or may be completely lost. The fields of vision may be concentrically contracted, irregularly limited, or may show quadrantal defects or abnormal scotomata. In consecutive atrophy the nasal portion of the field suffers first, and the colour-vision is always much affected. In primary atrophy the colour-sense is usually affected early, and the blindness may begin as a central scotoma. In all cases the condition tends to be steadily progressive, and to terminate in complete blindness, so that the prognosis is very bad. The interval between the onset and complete loss of sight depends, in consecutive atrophy, on the intensity of the original papillitis. It varies from three to four years, and, in tabes, from two months to seventeen years, the average time being about two years.

Treatment.—The treatment of optic atrophy is directed to its cause. When syphilis is present, anti-syphilitic treatment is employed. Strychnine given hypodermically (gr. $\frac{1}{30}$—$\frac{1}{15}$) once a day does good in some cases. Iron, arsenic, and phosphorus may be given as general tonics. Galvanism is sometimes of value, and some tabetic cases are said to have been improved by suspension.

THE THIRD NERVE.

The nucleus of the third nerve is situated in front of the aqueduct of Sylvius, and extends from the third ventricle to a point midway between the anterior and the posterior corpora quadrigemina. The nucleus is segmented into several groups, which have probably distinct functions. These groups have been named by the writer as follows (see Fig. 5):—

(*a*) A *median* group, from which fibres arise to enter both nerves. (*b*) An *anterior* group, lying on the inner and most anterior fibres of the posterior longitudinal fasciculus. (*c*) A *postero-external* group, lying on the most posterior and external fibres of this fasciculus. (*d*) An *external* group, lying partly external to and partly among the fibres of the fasciculus. (*e*) A *postero-internal* group (Edinger-Westphal nucleus), lying between the postero-external and the median groups.

The researches of Kahler and Pick, Henschen and Völckers, Allen Starr and others, seem to show that the centres for the various muscles are grouped within the nucleus in the following manner:—

Ciliary muscle.
Sphincter of iris. Rectus internus. Levator palpebræ superioris.
Rectus inferior. Rectus superior.
Obliquus inferior.
(Obliquus superior from the fourth nerve.)

The writer thinks the following association is the most probable:—The median group associated with the movement of convergence; the postero-internal with the ciliary muscle and the sphincter pupillæ; the postero-external with the elevators of the lid and the eyeball; and the anterior with the internal and inferior recti. The nucleus derives its blood supply from the perforating branches of the posterior cerebral arteries. It is worthy of note that the median and postero-internal groups (those supposed to innervate the ciliary muscle and sphincter iridis) are supplied from a set of vessels which do not enter the anterior and posterior groups of cells.

The nerve fibres (some of which may originate in the nucleus of the opposite side) pass forward and emerge in the interpeduncular space, near the middle line and internal to the motor tract from the cortex. The fibres nearest the middle line and most anterior at the point of emergence appear to be those for the sphincter pupillæ and ciliary muscle. The nerve passes along the outer side of the cavernous sinus before entering the orbit through the sphenoidal fissure.

THE FOURTH NERVE.

The nucleus of the fourth nerve lies in front of the aqueduct of Sylvius in the horizontal plane passing between the upper and lower corpora quadrigemina. After crossing to the opposite side in the valve of Vieussens, the nerve winds round the peduncle and reaches the superior oblique muscle by passing along the outer side of the cavernous sinus and through the sphenoidal fissure to the orbit.

THE SIXTH NERVE.

The sixth nerve arises from its nucleus in the floor of the fourth ventricle, passes almost directly forwards, through the pyramidal motor fibres, to emerge at the junction of the pons and medulla. It passes upwards in front of the pons, then along the outer side of the cavernous sinus to enter the orbit and supply the external rectus muscle. The sixth nucleus also supplies, either directly or indirectly, fibres to the internal rectus muscle of the opposite side. In this way it acts as a centre for conjugate deviation of both eyes to its own side. The nucleus has complex connections with the nuclei of the auditory nerve and the superior olivary body (see Fig. 6), which in all probability form paths for reflex impulses concerned in maintaining the equilibrium of the body.

Paralysis of the Oculo-motor Nerves.

Etiology and pathology.—All the nerves to the ocular muscles may be paralysed simultaneously by lesions in the orbit, sphenoidal fissure, or cavernous sinus—in the orbit, by injury, fracture of the bones forming its walls, acute or chronic cellulitis or periostitis, by the pressure of a tumour or exposure to cold; in the sphenoidal fissure, by fracture of the bones, periostitis, cellulitis, or tumour; in the cavernous sinus, by thrombosis, or the pressure of a dilated or aneurysmal carotid artery, or an adjacent tumour. At the base of the brain the nerves may be paralysed by meningitis,—simple, syphilitic, or tuberculous,—gumma or hæmorrhage, by the pressure of a tumour, or by infective or toxic neuritis, such as occurs in syphilis, diphtheria, influenza, diabetes, gout, alcoholism, and, though rarely, in lead poisoning.

The third nerve may be paralysed alone by a nuclear lesion, produced by hæmorrhage, by acute or chronic polio-encephalitis superior (Wernicke), by chronic degeneration of the cells, akin to that found in progressive muscular atrophy, or by the upward extension of that disease itself. Diphtheria or influenza may affect the nucleus, either alone or simultaneously with the nerves. Tabes or insular sclerosis may also damage the nucleus, and it is probably also affected in congenital oculo-facial palsy.

When the sixth nerve is paralysed alone, it may be affected in the orbit by injury or rheumatism, or involved in a localised meningitis at the base. When the lesion is intracerebral, it may be above, at, or below the nucleus:—

(*a*) *Supranuclear, i.e.* in the path between the cerebral cortex and the nucleus.

(*b*) *Nuclear* (in both these cases the lesion is generally a hæmorrhage, softening, or tumour).

(*c*) *Infranuclear.*—An infranuclear lesion within the pons may be one of the above mentioned, and generally affects the pyramidal motor fibres also.

The fourth nerve and nucleus are rarely affected alone, but tumours of the corpora quadrigemina, pineal gland, or anterior portion of the cerebellum, may affect the nucleus, and the nerve has been paralysed alone by localised basal meningitis. It may also be involved, in common with the other nerves, in the lesions already mentioned.

Symptoms.—Paralysis of the *third nerve* affects all the muscles of the eye, both internal and external, except those supplied by the fourth and sixth nerves (the superior oblique and external rectus). The symptoms are ptosis, *i.e.* drooping of the upper eyelid over the pupil, and inability to elevate it; divergent strabismus; diplopia; a moderately dilated and immobile pupil; loss of power of near vision in the affected eye. The eye cannot be moved upwards, downwards, or inwards, nor can it converge during attempted accommodation for a near object. It can be moved outwards by the sixth nerve, and rotated downwards and inwards by the fourth nerve.[1] The divergent strabismus may increase at a late date from contracture of the external rectus. The pupil does not contract either for light or for accommodation; but it can be dilated further by atropine. If the fourth and sixth nerves are also paralysed, the eye cannot be moved at all (complete ophthalmoplegia).

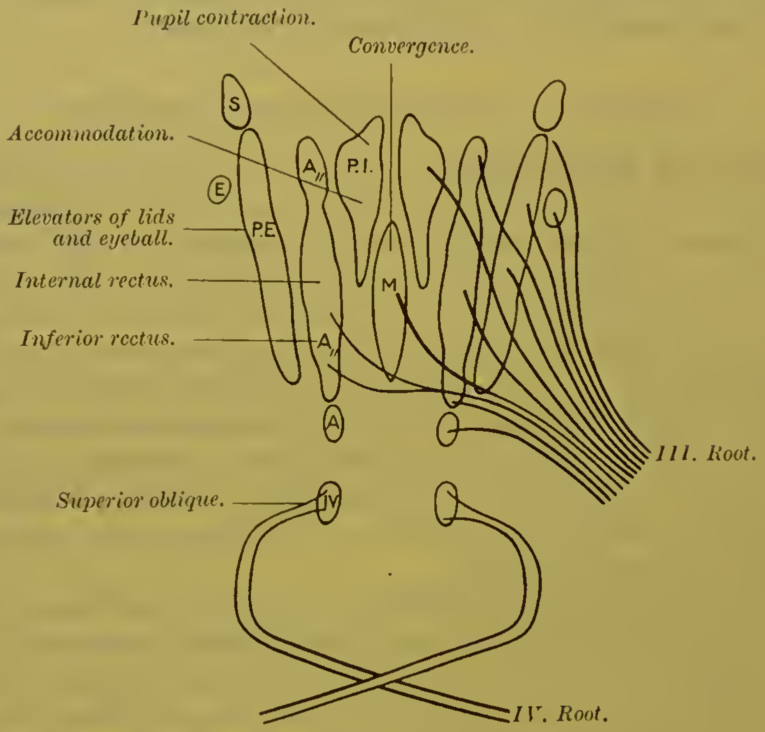

Fig. 5.—The segments of the third nucleus and their probable physiological relations. M, median group; A, anterior group; P.E., postero-external group; E, external group; P.I., postero-internal group; IV, the fourth nucleus.

Very frequently the paralysis of the third nerve is partial; thus the superior rectus and elevator of the lid may be affected alone or with one of the other recti, or all the muscles, with the exception of the levator, may be paralysed, or the internal muscles (sphincter pupillæ and ciliary muscle) may be alone paralysed (*ophthalmoplegia interna*), or may escape when the other muscles are involved. As a rule, an incomplete paralysis, especially if it is bilateral, is due to a lesion in the nucleus, and a complete paralysis to a lesion in the nerve; but to this rule there are numerous exceptions.

Besides sharing in the paralysis common to all the ocular muscles, the levator palpebræ may be affected alone. In congenital ptosis the lesion is probably a nuclear defect, and is bilateral and partial. The muscles of the eyeball are implicated to some extent, so that the globes cannot be fully rotated upwards, or there may be slight defects in some of the other movements. The condition is sometimes hereditary, and a form of slight double ptosis is common in the females of some families. To compensate for the paralysis of the levator, the frontalis over-acts, in order to raise the eyebrows and eyelids, giving the face a characteristic anxious expression.

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[1] The direction of rotation of the eyeball is stated in terms of the direction of rotation of the upper end of its vertical axis.

Morning ptosis is a condition allied to the above, which is present in sound sleepers, especially in weakly women. On waking in the morning, some difficulty is found in raising the eyelids, and in the case of debilitated women the lids may droop persistently for half an hour after rising. The condition passes off entirely during the day. Recovery is usually complete as strength is regained.

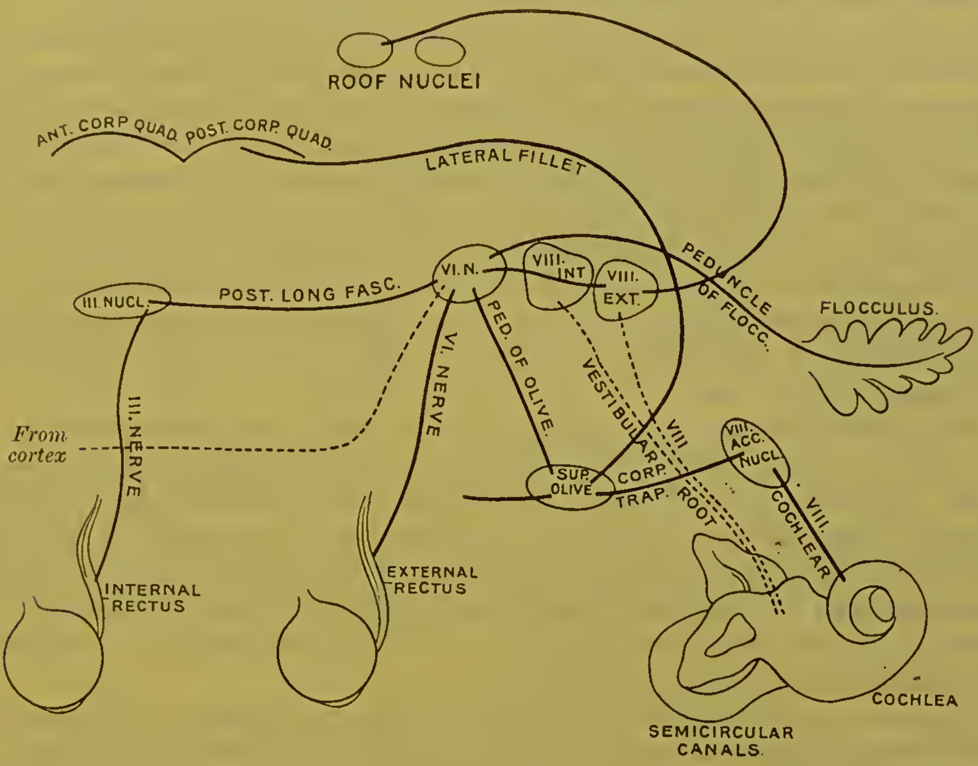


FIG. 6.—Diagram to show the relations of the sixth nucleus, and how it acts as the centre for lateral conjugate deviation of both eyes. VI. N., sixth nucleus; VIII. Int. and VIII. Ext., the internal and external nuclei of the auditory nerve; VIII. Acc. Nucl., the accessory acoustic nucleus or centre for the cochlear root; CORP. TRAP., the corpus trapezoideum.

Hysterical ptosis is not rare. It may be single or double, and is usually associated with spasm of the orbicularis, and sometimes with over-action of the frontalis. The spasm is seen when the patient is asked to look upwards, in which case the orbicularis contracts strongly, to prevent elevation of the lids.

Ptosis from paralysis of the sympathetic (Swanzy's sympathetic or pseudo-ptosis) is due to paralysis of the muscle of Müller at the back of the orbit. There is no loss of movement in the eyelids, but the palpebral fissure is narrowed, and the lid droops slightly on the affected side. Other symptoms of sympathetic paralysis are present. The face is reddened, the eye somewhat retracted (enophthalmos), its pupil is narrowed, and there is lachrymation on the affected side.

Reflex ptosis may be produced by irritation of the fifth nerve. It has followed division of the fifth nerve or the abstraction of a tooth.

In affections of the sixth nerve, when the lesion is supranuclear, there is usually at first conjugate deviation towards the side of the lesion. This, as a rule, soon disappears. It is, for obvious reasons, seldom an isolated symptom. A lesion of the nucleus itself produces loss of conjugate deviation of the two eyes towards the affected side. The eyes may show a slight convergence, and cannot be brought beyond the middle line towards the affected side. The head is usually slightly turned towards the paralysed side. Convergence for accommodation for near vision is retained. Bilateral paralysis of the sixth nuclei produces double loss of conjugate deviation, therefore the eyes cannot be moved towards either side, but yet they retain the power of converging for near vision. There is no strabismus.

Paralysis of the sixth nerve produces a convergent strabismus—the affected eye cannot be moved beyond the middle line. There is secondary over-action of the opposite internal rectus, so that when the patient looks towards the paralysed side, the opposite eye turns too far inwards. Diplopia is always present, and the distance between the images increases on looking toward the paralysed side. The erroneous projection of the image leads to a feeling of giddiness. The movements of the opposite eye are not impaired. If the lesion is in the pons, the seventh nerve of the same side, or the tongue, arm, and leg of the opposite side, may be paralysed. If at the base of the brain, near the cavernous sinus, the other ocular nerves may be involved.

The superior oblique muscle supplied by the fourth nerve rotates the eye, so that the upper end of its vertical axis rotates inwards and downwards. It also depresses and slightly everts the eye. When the muscle is paralysed, these actions are lost. The defective movement is best seen when the eye is depressed beyond the horizontal plane. The affected eye is more inverted but less depressed than its fellow; its motion inwards and downwards is lost; diplopia is present on looking downwards, and especially downwards and outwards.

There are certain general symptoms which are common to all paralyses of the external ocular muscles. They may be briefly stated as follows :—

Primary deviation of the affected eye.—This is due to the unopposed action of the antagonists of the paralysed muscle, and must therefore be in a direction opposite to its action.

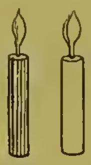
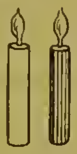
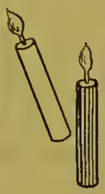
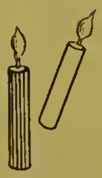
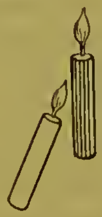
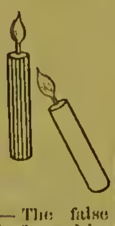
Secondary deviation of the sound eye.—This is always greatest on attempting to look towards the paralysed side, owing to the over-stimulation of the sound muscle in the healthy eye, in the effort to use the paralysed one of the affected eye.

Diplopia and erroneous projections of object seen by the paralysed eye.—The diplopia is due to the formation of images on portions of the retina which do not correspond, owing to the primary deviation, and to the absence of muscular movement on stimulation of the paralysed muscles of the affected eye. The false image is always projected in the direction in which the paralysed muscle would normally move the eye. The distance between the images is greatest when the movement of the eye is in the direction of the action of the paralysed muscle. They approach each other, and finally fuse when the eye moves in the opposite direction. It will be seen, therefore, that the position of the false image can always be deduced from the action of the paralysed muscle, and, conversely, the muscle which is paralysed can be determined by the position of the false image.

The position of the face is altered so as to avoid diplopia by rendering the axes of the eyes as nearly parallel as possible. It is therefore turned in the direction of the action of the paralysed muscle, so as to compensate for the lack of movement in the latter.

Vertigo is frequently present, and is due to the erroneous projection of objects in the field of vision, causing want of co-ordination between sight and the other senses by which we gain a knowledge of our position with regard to other objects.

The following scheme indicates briefly the normal action of the various muscles, the position of the eye, the face, and the false image seen in their several paralyses. In each case the muscles of the right eye are supposed to be affected.

Muscle.	Normal Movement.	False Image.	Position in Paralysis.	Double Vision.
External rectus.	Outwards.		Eyes turned inwards. Inward secondary deviation of sound eye on looking to right. Face turned to right.	Diplopia (homonymous) on right half of field.
Internal rectus.	Inwards.		Eye turned outwards. Outward secondary deviation of sound eye. Face turned to left.	Diplopia (crossed) on left half of field. Diplopia greatest to left.
Superior rectus.	Upwards. Inwards. Inward rotation of vertical axis.		Eye lower. ,, outwards. ,, outward rotation. Upward secondary deviation of sound eye. Face turned upwards.	Diplopia (crossed) in upper field. Most marked as eyes move upwards.
Inferior oblique.	Upwards. Outwards. Outward rotation of vertical axis.		Eye lower. ,, inwards. ,, inward rotation. Face turned upwards.	Diplopia (homonymous) in upper field. Most marked when eyes move up and out.
Inferior rectus.	Downwards. Inwards. Outward rotation of vertical axis.		Eye higher. ,, outwards. ,, inward rotation. Downward secondary deviation of sound eye. Face turned down and to the right.	Diplopia (crossed) in lower field. Most marked when eyes move downwards.
Superior oblique.	Downwards. Outwards. Inward rotation of vertical axis.	 FIG. 7.—The false image is formed by the unshaded candle.	Eye higher. ,, inwards. Slight outward rotation. Face turned downwards.	Diplopia (homonymous). No diplopia above horizontal plane. Vertical distance greatest when eyes turned down and in.

Treatment.—The treatment of ocular paralysis resolves itself into treatment of the cause, and local treatment of the paralysed muscles. Syphilis or rheumatism should be treated in the usual manner, the general health attended to, and iron or arsenic and strychnine given when necessary. In cases of local neuritis, mercury is the most useful drug.

The local applications vary according to the stage of the disease. In recent acute cases blisters may be applied behind the ear, on the occiput, or on the temple when the disease is orbital. Acute rheumatic cases may be relieved by repeated hot fomentations over the eye and temple. In more chronic cases, iron, arsenic, and small doses of strychnine may be tried. Electricity is of very little use in those cases. It should never be applied to the conjunctiva, for even although the pain of the application may be deadened by cocaine, there is great risk of injuring the structures of the eye. The voltaic current may be applied to the muscles through the closed eyelid, the physician using his own wetted finger as an electrode in order to estimate the strength of the current, and the eye being turned away, to expose as much as possible of the paralysed muscle. The faradic current cannot be used in this way, as it does not pass through the eyelid.

A prism may be used over the affected eye to counteract the diplopia. It should not be strong enough to fuse the images entirely, as it is desirable that there should be some stimulus to action of the paralysed muscles. A weaker prism should be used daily as a gymnastic exercise. The giddiness produced by the erroneous projection may be obviated by the use of an opaque glass over the affected eye; but this should only be used as a last resource, as it does away with any stimulus to movement of the weak muscles. When contracture has occurred, the tendon of the muscle may be divided and allowed to attach itself farther back.

The treatment of ptosis is similar to that of the other muscles, except that in neurasthenia local stimulating liniments may be used. These cases are usually very amenable to treatment by tonics, such as strychnine or quinine. The hysterical forms do not yield so readily, and it may be necessary to apply blisters to the temple, or in unilateral cases to tie up the sound eye.

SPASM OF THE OCULAR MUSCLES.

In some forms of cerebral disease, associated spasm of the ocular muscles is found, producing conjugate deviation. When the lesion is destructive the deviation is towards the diseased side, since the action of the healthy hemisphere is unopposed; but when the lesion is irritative the eyes look away from the side of the lesion.

Lesions at the base of the brain, of which meningitis is the most common, may produce irregular spasm in one or more muscles. Convergent squint is very common in basal meningitis; but single muscles may also be affected, very often intermittently. In some cases of chorea, transient spasm of some of the ocular muscles may be present, and diplopia may result. In hysteria, spasm of the ocular muscles may be seen. A high degree of convergent strabismus may be present, the eyes may be turned up and in, or ptosis or apparent paralysis of the abducens may appear. Divergent strabismus is never found in hysteria.

During paroxysmal convulsive attacks the eyes deviate towards the side on which the convulsion is greatest, and, after the convulsion is over, they usually deviate slightly from the convulsed side, since they share in the weakness of the affected muscles. In some cases (Gowers) a paroxysmal tonic spasm appears, limited to one ocular muscle, and lasting a brief space, but producing no symptoms beyond some diplopia and giddiness. Sometimes clonic spasm of the orbicularis accompanies the condition, and after the attack the eyelid droops on the affected side. Irritation of the fifth nerve has been known to produce spasm of the levator palpebræ, sometimes synchronous with movements of the maxilla. It is probably a reflex or associated movement. Gowers suggests that the levator fibres of the third nerve are derived from the motor division of the fifth nucleus, but this is not likely.

The spasm found in squint due to errors of refraction, or in the secondary deviation in paralysis, need not be considered here.

NYSTAGMUS.

This is a rapid involuntary movement of the eyeballs, usually bilateral and equal in each eye. The direction is most commonly lateral, but it may be vertical or even rotatory. In unilateral cases it is, as a rule, vertical. It may be continuous or intermittent, the latter form being due to paresis of one of the ocular muscles—the nystagmus only occurring when the weak muscle is put in action.

The character of the movement varies in extent and rapidity. It may occur sixty times in the minute, or be too rapid to be counted, and the extent varies from 10 mm. to a movement so slight as to be only visible on ophthalmoscopic examination. It consists of a rapid rhythmic, usually quite regular, movement in one direction, with a slow movement back again. In some cases the rate of movement is slow while the eye is at rest, but rapid when the eye is moved voluntarily.

It is due to many causes—local disease of the eye causing impairment of sight, such as opacities in the lens, degenerative or inflammatory diseases of the retina or choroid in childhood. These causes are rarely sufficient in adult life. It is found in rickets, sometimes associated with head nodding. In albinism, nystagmus is common, and it also occurs in miners and others who work in a bad light with the eyes constantly turned in one direction. It is frequently present in lesions in the pons and optic thalamus and in some chronic degenerative nervous diseases, such as hereditary (Friedreich's) ataxia and insular sclerosis, but is not present in locomotor ataxia or paralysis agitans. It has been observed in alcoholic neuritis. It is found in many forms of cerebral disease, most commonly in meningitis, hæmorrhage and tumours, and is specially common in tumours of the cerebellum and pons Varolii. Gowers states that it is more likely to be found in cerebral disease if optic neuritis is also present. In chronic otitis pressure on the middle ear may cause nystagmus, and it is also present in health during rapid rotation of the body, in both cases associated with vertigo. It ceases entirely during sleep. The patient, as a rule, is unconscious of the condition, except when it is developed in adult life, when he may be aware of the apparent movement of objects in the field of vision.

As a diagnostic or localising symptom, nystagmus has hitherto proved of little value, except in distinguishing insular sclerosis from hysteria. In the functional disease nystagmus is never present.

RECURRENT OCULO-MOTOR PARALYSIS.

This is a rare affection (often termed migraine ophthalmoplégique) in which the ocular muscles of one eye are subject to transitory paralysis at periodic intervals. It begins in early childhood, as a rule, but may first appear in old age. The female sex is affected more commonly than the male, and the left nerve more frequently than the right. The exciting cause is unknown, but a neurotic tendency may predispose to it, and it sometimes comes on in those who have been subject to migraine previously. The attacks come on periodically, always affecting the same side and lasting a variable time, from a few days to months. They are accompanied by severe pain in the eye, the forehead, or temple on the affected side, by nausea, and sometimes vomiting and slight pyrexia. The headache is migrainous in character, and may persist for days or weeks. Occasionally the eyelids are swelled, there is a purulent discharge from the eye, and anæsthesia is present in the distribution of the ophthalmic branch of the fifth. Facial paralysis, and similar recurring attacks affecting several cranial nerves, or the sixth or fifth alone, have been described. In the intervals the muscles may be quite normal, or they may be slightly paretic.

The pathology of the condition is not definitely known. It is evidently a basal lesion, most often meningitic. In the three recorded cases on which an autopsy was performed, one was found to show meningitis round the third nerve at its exit from the pons, and the other two showed a small new growth in the same situation. The writer has seen a case due to a slight localised basal meningitis implicating the third nerve. Senator considers it is a reflex paralysis. Charcot regarded it as closely allied to migraine, and called it migraine ophthalmoplégique. The prognosis is good as regards life, unless the disease be due to an organic lesion. As regards duration it is not so favourable. In some cases arrest may occur, but it very commonly lasts for life. Its treatment is similar to that employed in cases of migraine, prolonged treatment with the bromides being of most use.

It is best treated as migraine; no local treatment is of any use.

INTERNAL MUSCLES OF THE EYE.

The sphincter iridis, which contracts the pupil, is innervated by one of the most anterior segments of the nucleus of the third nerve. Its special fibres are situated in the anterior and innermost part of the emerging root. The dilator muscle of the iris is supplied by the sympathetic nerve. Its centre appears to lie below the anterior corpora quadrigemina, and to the outer side of the third nucleus. From this centre the fibres descend to the cilio-spinal region in the lowest cervical portion of the cord, emerge by the anterior roots of the first dorsal nerve (Oppenheim), to again ascend in the sympathetic through the carotid and cavernous plexuses to the nasal branch of the first division of the fifth nerve, whence they pass to the lenticular ganglion by the long sensory root. The ciliary muscle (for accommodation) is supplied through the short ciliary nerves of the lenticular ganglion. The centre is probably the single median group in the anterior part of the oculo-motor nucleus. [According to Heddæus, the nucleus sends special fibres, which subserve pupillary contraction for accommodation to join the fibres from the sphincter nucleus, the two sets of fibres thus forming the iris branch of the third nerve. It seems more likely that contraction for accommodation and convergence is produced by a direct action of the centre for these movements on the sphincter centre.]

The average size of the pupil is 4 mm., but it is in a state of constant variation. It oscillates synchronously with the pulse and respiration, is contracted in the morning, in old age, and in deep sleep, and dilated in the afternoon, in childhood, and under the influence of cutaneous stimulation, co-ordinated muscular action, or of emotion. There are in addition reflex and associated movements, namely, contraction to light, dilatation in darkness, and the contraction associated with accommodation and convergence. There is also a contraction, independent of light, convergence, or accommodation, when the attention is fixed on a particular object.

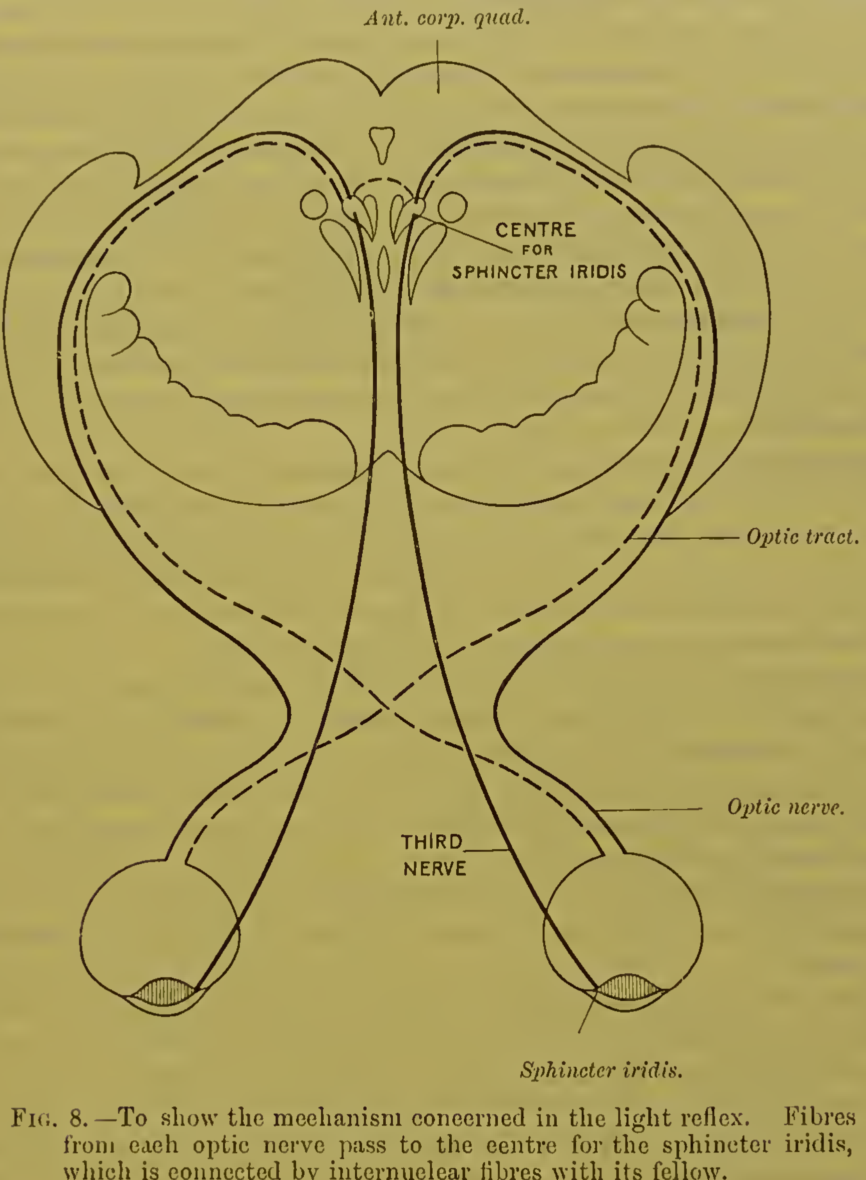


FIG. 8.—To show the mechanism concerned in the light reflex. Fibres from each optic nerve pass to the centre for the sphincter iridis, which is connected by internuclear fibres with its fellow.

Light thrown into an eye produces a direct reflex contraction in that eye, and an equal consensual contraction in the other. When both eyes are stimulated by light, the resulting contraction is greater than when one alone is illuminated. The reflex arc consists of—retina, optic nerve, chiasma, both optic tracts and special fibres from these passing through both anterior corpora quadrigemina, and ending in relation to the sphincter iridis centre in both oculo-motor nuclei, and, finally, the fibres of the third nerve supplying the muscular fibres of the iris. The existence of a decussation of the light fibres in the chiasma is proved by the persistence of both direct and consensual contraction of the pupil in hemianopia due to a lesion of the optic tract; while the presence of some further communication between the nuclei for the sphincter of the iris is proved by a case, quoted by Weir Mitchell, and by an experiment of Bernheimer in the monkey, in which the centre of the chiasma had been completely cut across, and yet both direct and consensual reaction were preserved. The reflex mechanism is made evident by the accompanying diagram (Fig. 8), which illustrates the result of Bernheimer's observations.

The associated contraction of the pupil occurs during convergence and accommodation. Convergence seems to be the controlling action, since it is invariably accompanied by pupillary contraction, whereas accommodation may occur alone. There is some reason to believe that there are separate centres for convergence contraction and light contraction. Dilatation of the pupil is under the control of the sympathetic. The influence is tonic, becoming apparent when the action of the third is in abeyance, as when the eye is in darkness or fixed on a distant object. The tonic action of the sympathetic may be augmented, and wide dilatation produced, by irritation of the sympathetic, by sensory stimuli, co-ordinated muscular actions, and strong emotion.

Symptoms.—**Ophthalmoplegia interna.**—This denotes complete paralysis of the internal muscles of the eye. The pupil is dilated and immobile, showing no reaction to light or for accommodation, but dilating still further under the action of atropine. The power of accommodation is lost. The lesion affects either the anterior fibres of the emerging root of the third nerve, or the anterior part of its nucleus.

Cycloplegia.—In paralysis of the ciliary muscle there is no power of accommodation, so that, while distant vision is perfect, near vision is lost, but can be restored by the use of convex lenses. When cycloplegia is unilateral, it is due to a peripheral lesion. A nuclear lesion always causes bilateral paralysis of accommodation. The condition is usually due either to tabes or to diphtheria.

Iridoplegia.—With paralysis of the sphincter iridis the pupil is dilated and immobile, and does not contract either for light, accommodation, or convergence.

Reflex iridoplegia.—In this condition, commonly known as the Argyll Robertson symptom, the pupil, while contracting normally for convergence and accommodation, does not contract under the stimulus of light. It is usually bilateral and associated with myosis, and occurs commonly in tabes and general paralysis. When bilateral it is probably due to an interruption in the connection between the terminations of the light fibres of the optic tracts in the anterior corpora quadrigemina and the centre in the third nucleus for the sphincter iridis, *i.e.* between the central ends of the sensory and motor segments of the reflex arc (see Fig. 8). When the iridoplegia is unilateral,—a rare occurrence,—and unassociated with cycloplegia, it is more difficult to determine the seat of the lesion.

Association iridoplegia is the condition in which the contraction of the pupil during attempted accommodation is lost, although the pupil may contract to light or during convergence. The ciliary muscle may or may not be paralysed. The lesion in such a case, according to Heddæus, is in the fibres from the accommodation nucleus which control the sphincter muscle. It may, however, divide the connection between the accommodation and sphincter centres.

Cutaneous iridoplegia.—In this condition the pupil, which is usually contracted, does not dilate when the skin is stimulated, although all the other actions of the pupil may be present. The lesion in such a case is in the cervical sympathetic, the cervical cord, or the medulla. It is always necessary to test whether cutaneous sensibility is present, as its absence may account for the loss of the reflex.

Lesions in the course of the centripetal part of the reflex arc may affect the actions of the pupil in various ways. When the lesion is in one optic nerve, there is no direct reflex contraction of the pupil for light in the affected eye, nor consensual contraction in the other; but when light is admitted to the sound eye, both pupils contract equally. Convergence and accommodation are normal. When the lesion is in the optic tract, the pupils of both eyes will contract both directly and consensually if light is thrown on to the seeing halves of the retina; but if light is thrown on to the blind halves, no reaction will be obtained in either eye.

Mydriasis, or abnormal dilatation of the pupil, may be due to general causes, such as anæmia, neurasthenia, or the late stages of the stupor caused by alcohol or chloroform. It may be due to some local cause, such as refractive error, glaucoma, atrophy of the optic nerve, or the instillation of mydriatics, most commonly atropine. Strong emotion will also produce wide dilatation. Paralytic mydriasis is seen in paralysis of the third nerve or nucleus, and sometimes in the early stages of ataxia. Irritation mydriasis is the dilatation of the pupil due to irritation of the cervical portion of the cord, in meningitis and tumours.

Myosis, or contraction of the pupil, may be produced by local lesions, such as refractive error, iritis, simple congestion of the iris, or by the instillation of myotics. It is also present in poisoning by opium, alcohol, or chloroform, in uræmia, during epileptic seizures, in the course of the specific fevers, and sometimes in the venous obstruction due to mitral disease. Irritation myosis may be due to meningitis, sinus thrombosis, tumours or abscess of the brain in their early stages, or to small hæmorrhages into the cerebellum. It is sometimes seen in cerebral apoplexy, especially at the beginning of the attack, and in hæmorrhage within or pressing upon the pons. Paralytic (spinal) myosis is seen in some chronic progressive nervous diseases, such as tabes, bulbar paralysis, insular sclerosis, and in some forms of general paralysis.

CONDITION OF THE PUPILS IN VARIOUS DISEASES.

In *tabes*, one of the earliest signs is inequality of the pupils. They may also be irregular in shape, sometimes elliptical. In size they vary, from a high degree of contraction to a moderate degree of dilatation, myosis being the most common condition. They respond readily to myotics by extreme contraction, but are not fully dilated by mydriatics. Irritation of the third nerve may produce spastic myosis, and its paralysis results in paralytic mydriasis. Spastic mydriasis, due to irritation of the dilator fibres in the posterior columns in the cervical cord, is also sometimes seen. Reflex iridoplegia is present in about one-half of the cases. It is usually bilateral, but sometimes, though rarely, unilateral. It may be the only eye symptom, or it may be associated with myosis, mydriasis, or inequality of the pupil. Association iridoplegia usually appears later in the disease, and the response to cutaneous stimulation may also fail. It is very rare for the loss of pupillary contraction for convergence and accommodation to occur without failure of the light reflex.

In *general paralysis* the pupils may be unequal, irregular, contracted, or dilated. Loss of reaction to cutaneous stimulation is often the first eye symptom to appear, and may be due simply to loss of cutaneous sensibility. It may be succeeded by reflex iridoplegia, partial or complete, associative iridoplegia, and cycloplegia, so that complete internal ophthalmoplegia may thus be present. In some cases the dilatation on stimulation of the skin persists until late in the disease.

In *epilepsy* there may be marked myosis during the seizures, and the pupils are frequently unequal, especially when a localised intracranial lesion is present. The inequality may not be apparent, except when the eye is faintly illuminated.

In *insanity* inequality of the pupils may be a marked premonitory symptom.

Hippus (the rhythmical contraction and dilatation of the pupil, present normally for a few seconds after light stimulus to the retina) is seen in some nervous diseases. It has been observed in multiple sclerosis, and in neurasthenia (Damsch), in epilepsy, acute meningitis, and oculo-motor paralysis. Dercum records two cases, one of recurrent mania, and one of grave hysteria, in which the condition was very marked.

THE FACIAL NERVE.

The seventh nerve rises from a nucleus situated in the lower part of the pons, nearly midway between its anterior surface and the floor of the fourth ventricle, and slightly internal to the ascending root of the fifth nerve. From this point the root passes backwards and inwards, curves round the nucleus of the sixth nerve, forming a loop (the knee of the facial), and is then directed forwards and outwards, to emerge at the lower border of the pons external to the sixth nerve, and in close proximity to the auditory nerve. The further course of the facial nerve may be divided into four parts: first, the portion between the pons and the geniculate ganglion; second, between the geniculate ganglion and the origin of the chorda tympani; third, between the origin of the chorda tympani and the stylo-mastoid foramen; and, lastly, the peripheral distribution of the nerve external to the stylo-mastoid foramen. In the first part it lies in the internal auditory meatus, accompanied by the auditory nerve and the portio-intermedia of Wrisberg, a nerve whose functions are not yet known. In its second part the facial nerve has received the chorda tympani, and lies in close relation to the middle ear, giving off a small branch to supply the stapedius muscle. In the third part the nerve runs alone through the mastoid bone, and terminates in the fourth part by dividing into three great branches, temporo-facial, cervico-facial, and posterior-auricular, supplying all the muscles of expression, and, in addition, the stylo-hyoid and posterior belly of the digastric. There is some reason to believe that the orbicularis palpebrarum is innervated from a centre in the neighbourhood of the third nucleus, and it is considered by many that the muscles of the lips are supplied from the hypoglossal nucleus. It is not known, however, how the fibres from these two sources pass to the facial nucleus.

FACIAL PARALYSIS.

Etiology and pathology.—The lesions which produce facial paralysis may be classified according to their position in the course of the path from the cerebral cortex to the periphery. They may occur in the intracranial, the intrapetrous, or the peripheral or extracranial portion. Within the cerebrum, and in the crura and upper pons, the lesions which may produce facial paralysis are hæmorrhage, softening (from endarteritis or embolism), tumours, syphilis, abscess, sclerosis, and tuberculous disease. In the lower pons any of these lesions may affect the nerve, and, in addition, nerve or nucleus may be affected by tabes, amyotrophic lateral sclerosis, bulbar paralysis, and polio-encephalitis superior. Cerebellar hæmorrhage occurring at birth may also affect the nerve in this situation. At the base of the brain the nerve may be involved in meningitis, or compressed by

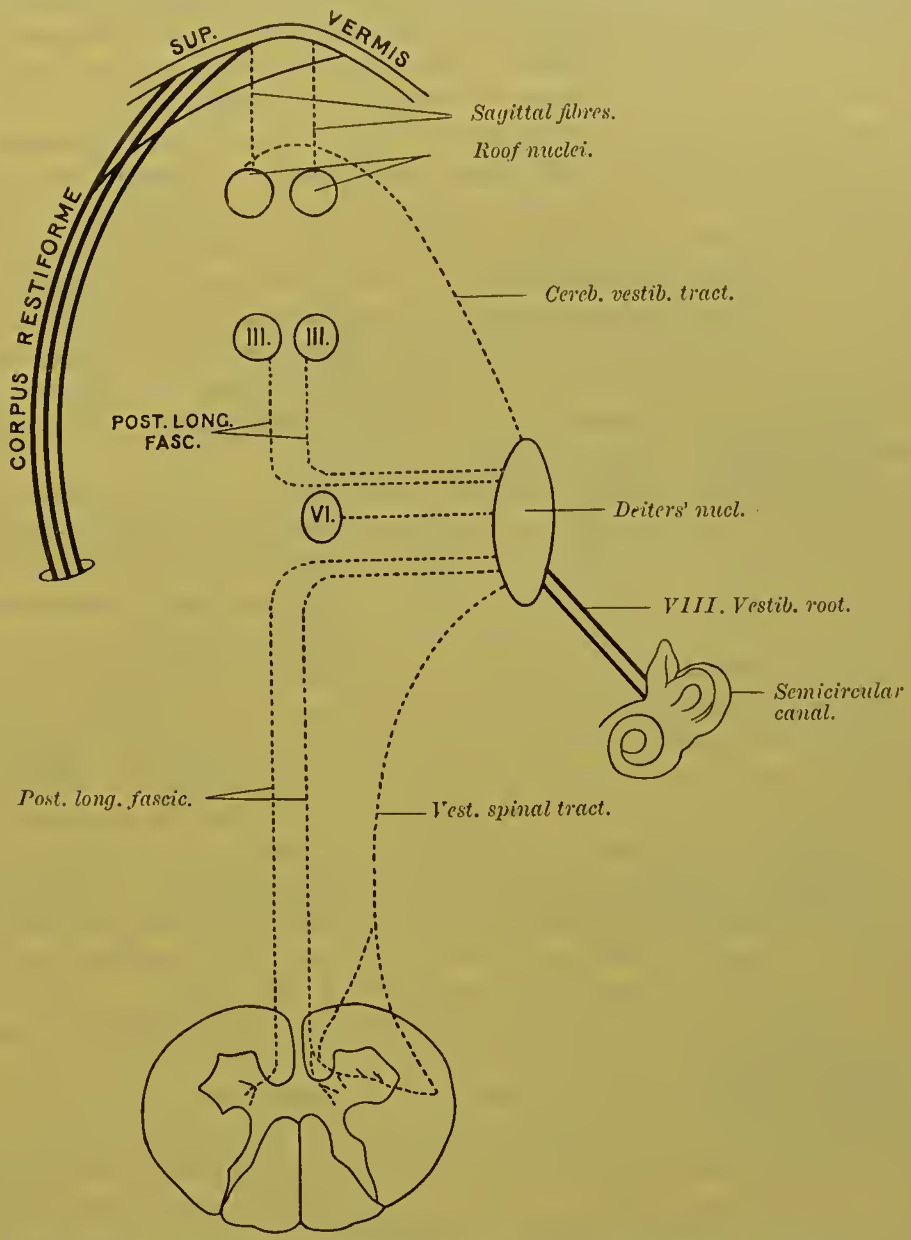


FIG. 9.—Diagram to show how the middle lobe of the cerebellum and the nucleus of the vestibular root of the eighth nerve may influence the third and sixth nuclei, and also the movements of the same side of the body.

tumour or by aneurysm. Within the petrous bone, the common lesions which involve the facial nerve are periostitis, caries or necrosis of the bone, suppurative or catarrhal middle ear disease, fractures of the base, thrombosis during the puerperium, haemorrhage into the nerve, and, lastly, neuritis, either infective or toxic.

In its extracranial course the nerve may be affected as a whole, or the paralysis may be limited to some branches, according to the site and character of the lesion. The most common general cause is rheumatic neuritis. This may appear at any age, but is rare before 10 and after 50. Some very intractable cases come on late in life, however, between 74 and 77. It is more common in males than in females, and occurs more frequently in winter than at any other season. Neuritis may also be due to alcoholism, diphtheria, diabetes, tetanus, puerperal disturbances, or to poisoning with lead or mercury. In the acute specific diseases, scarlatina, typhoid fever, diphtheria, etc., the neuritis may be primary or secondary. The nerve may be directly affected by extension of the inflammation in parotitis and tonsillitis, or by the irritation of a wisdom-tooth, and may be destroyed in whole or in part by an injury, such as a gunshot wound, a stab, or a blow on the side of the face, or by tumour or operation in this neighbourhood. The application of forceps during birth may paralyse the nerve.

Pathology.—Since facial paralysis is never in itself fatal, the pathology of the condition cannot be well known. No reason is known why this nerve should be specially affected, nor, in most cases, what elements are first or chiefly involved. The rheumatic neuritis was formerly said to consist of an infiltration and inflammation of the nerve sheaths, *i.e.* it was an interstitial or peri-neuritis. Minkowski and Déjerine, however, in cases of rheumatic neuritis, have found that there was no peri-neuritis present, but that the changes in the nerve were purely degenerative. In infective and toxic cases, *e.g.* diphtheria, alcoholism, the neuritis is parenchymatous. Hubschman thinks that the so-called rheumatic form may be an infective disease. In syphilitic cases the nerve shows the small round-celled infiltration characteristic of the disease. The late over-action and spasm are due to the altered condition of the nerve cells of the nucleus. Their resistance is lowered, and they discharge spontaneously. The contractures always coincide with the return of some degree of power, and never appear in muscles that are completely and permanently paralysed.

Symptoms.—**Unilateral.**—In a typical case of paralysis affecting all the branches of one facial nerve, there is complete loss of voluntary movement in the muscles of expression on the corresponding side of the face, so that that half of the face is immobile, smooth, and mask-like. On the affected side, the wrinkles on the forehead and the naso-labial fold are obliterated. The mouth and the tip of the nose are drawn to the sound side by the unopposed action of the healthy muscles, and on the affected side the corner of the mouth droops and the nostril is flattened. These appearances are naturally more visible in the strongly-marked face of adult life, when the tissues have lost some of their elasticity, than during childhood. The eye on the affected side is somewhat more widely opened than on the sound side, and may water slightly, since the weakened orbicularis cannot keep the punctum lachrymale apposed to the eyeball, nor can the eye be closely shut. When an attempt is made to close the eye, there is a rotation upwards of the eyeball so that the cornea disappears below the upper lid.

Frowning or wrinkling the skin of the forehead on the affected side is impossible, as the frontalis and corrugator are both paralysed. The muscles of the lips and cheeks are also markedly involved, so that when the patient tries to show his gums the mouth is drawn over to the sound side, and when he attempts to whistle the air escapes on the paralysed side. Food tends to accumulate between the cheek and the teeth, owing to paralysis of the buccinator, and fluids may escape between the imperfectly apposed lips. In speaking, the labials are not clearly pronounced. When the tongue is protruded, it shows an apparent deviation to the affected side, but this is merely owing to the displacement of the lips, which are pulled over to the sound side. These symptoms may appear rapidly, sometimes in the course of a night, and without warning, or they may be preceded by neuralgic pains near the ear, in the lower jaw, or the side of the head. Giddiness and deafness are sometimes present, and there is sometimes in front of the ear a swelling due to local cellulitis.

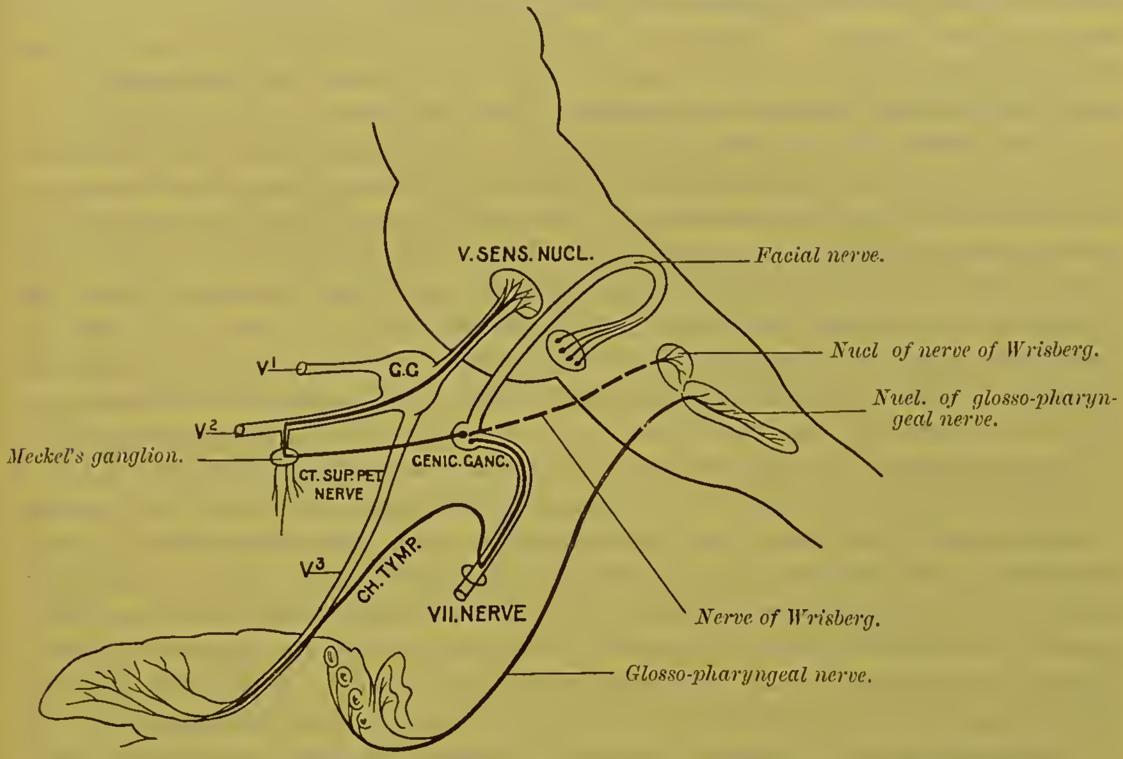


FIG. 10.—Diagram to show the paths of the nerves of taste. CH. TYMP., the chorda tympani from the anterior two-thirds of the tongue to the geniculate ganglion, whence its central path passes by—(*a*) the nerve of Wrisberg and (*b*) by the great superficial petrosal nerve (GT. SUP. PET. NERVE), Meckel's ganglion, the second division of the fifth nerve, to centres in medulla and pons.

The symptoms vary a little according to the site of the lesion. If the nerve is injured between the geniculate ganglion and the stylo-mastoid foramen, there may be, in addition to the other symptoms, loss of taste in the anterior two-thirds of the tongue on the affected side, owing to paralysis of the chorda tympani. If the nerve to the stapedius be involved, there is hyperakusis (increased sensitiveness to sound), as estimated by Galton's whistle, but very frequently there is more or less complete deafness. Abnormal sensations of taste may appear, due to implication of the middle ear, the tympanic plexus, and Jacobson's nerve by the disease. When the lesion is situated higher than the geniculate ganglion, between it and the pons, the auditory nerve is almost invariably affected, and nerve deafness thus produced. In such a case, there is no loss of taste, however, as the chorda tympani fibres only join the facial nerve at the geniculate ganglion. When the lesion is at the base, some of the other cranial nerves are also usually affected, most frequently the sixth and the eighth.

The evidence as to involvement of the palatal muscles is very conflicting, but seems on the whole to indicate that they are not affected in pure facial paralysis (Hughlings Jackson, Gowers). When the lesion is in the nucleus of the seventh nerve, the lower face muscles are first and most severely affected, whereas the orbicularis palpebrarum may be to some extent spared. There is nearly always paralysis of some of the other cranial nerves on the same, or on both sides. In all nuclear and infranuclear lesions the reflex action of the facial muscles is abolished.

The result of a supranuclear lesion in the pons, involving the tract from the cerebrum to the facial nucleus, varies according to whether it is above or below the decussation of the facial fibres. When it is below this point, there is alternate paralysis of the face and limbs, an injury in this region being rarely limited to one set of motor fibres, whereas when the lesion is above the decussation, the face and limbs of the *same* side are paralysed, *i.e.* the side opposite the lesion. It is said (Gowers) that paralysis of the orbicularis palpebrarum alone indicates a lesion destroying the fibres between the third and facial nuclei, and that isolated paralysis of the orbicularis oris shows that the lesion is near the hypoglossal nucleus.

Lesions in the cortex, or interrupting the facial fibres in the internal capsule or crus, all cause paralysis of the face and limbs on the side opposite the lesion. When the cortex is affected the paralysis is seldom complete, since the orbicularis palpebrarum and the frontalis muscles are partially spared. When the crus is the site of the lesion the oculo-motor nerve is very often involved, and ophthalmoplegia is present on the same side as the lesion, the face and limbs of the opposite side being also paralysed. In all lesions above the nucleus the reflex action of the facial muscles is intact.

The electrical reactions will be discussed under Prognosis. In slight cases of facial paralysis the muscles rapidly regain their tone, and the face becomes normal, but in old-standing cases, over-action and contracture appear in the affected muscles, mainly in the zygomatici. The angle of the mouth is then pulled over to the paralysed side, and the naso-labial fold becomes exaggerated, so that it may be deeper than that of the sound side, and it may be difficult to tell which is the paralysed half of the face. The condition may persist for years, or become permanent. It rarely passes off entirely.

In cases where the orbicularis palpebrarum is completely paralysed, the cornea is frequently injured by exposure, and by foreign bodies which enter the unprotected eye. Ulceration of the cornea and conjunctivitis may thus be produced, the former being very rare. Sensation is unaffected unless the fifth nerve is implicated, and no trophic changes occur, except, in old cases, wasting of the paralysed muscles from disuse.

Bilateral.—Bilateral facial palsy may be produced by two separate lesions in the cerebrum, somewhere in the course of the facial fibres, or by a single lesion in the pons, involving the decussation of the fibres, or attacking the nuclei (progressive bulbar paralysis). Hæmorrhage, softening, syphilis, and tumour may affect the nucleus or the nerve within the pons, and meningitis (syphilitic or tuberculous), disease of the petrous bone, the periosteum, or the middle ear of both sides, may affect the nerve in its extracerebral course. A fall or a blow causing a double fracture has been known to cause bilateral facial paralysis; and both nerves may be affected by acute multiple neuritis, due to toxic conditions, and by diphtheritic paralysis, or by the pressure of forceps during instrumental delivery.

The appearance of such a case is very typical. There is absolutely no power of expression. The whole face is smooth and unwrinkled, like a mask, the under lip droops a little; and this, combined with the loss of movement in the eyebrows, gives a peculiar sullen expression to the face. The symptoms, course, and prognosis of the disease are similar to those given under the description of unilateral paralysis.

Diagnosis.—The effects of paralysis of the face are so striking that in the early stage it is not difficult to diagnose. It should always be borne in mind, however, that the statements of the patient and his friends may be unintentionally misleading, as they often regard the smooth, unwrinkled side of the face as the normal one. It is not so easy to decide which half of the face is paralysed later in the disease, when contracture and over-action are present, and mask the paralysis. In such cases, attention must be paid to the movements of the face, and it will be seen that, on a slight attempt at motion, the paralysed half of the face moves most markedly, but when the amount of movement attempted is great the sound side is much more active.

The site of the lesion may in many cases be determined by the amount of paralysis and the associated symptoms. When the disease is supra-nuclear, the orbicularis is not completely paralysed, and paralyses are present in other parts. There is more power of emotional movement of the face than when the lesion is nuclear and infranuclear. Reflex and electrical reaction are not affected. In nuclear and infranuclear lesions the paralysis of movement, whether voluntary or emotional, is complete, the reflexes are absent, and in moderate or severe cases the reaction of degeneration appears. It must be borne in mind that a lesion of the nerve may be partial, so that the electrical reactions may not be much impaired. When the lesion is in the lowest part of the Fallopian canal, or extracranial, the facial nerve is paralysed alone, and taste is not affected; whereas, if it is between the geniculate ganglion and the origin of the chorda tympani, the sense of taste is lost on the anterior two-thirds of the tongue of that side. When the lesion is in the internal auditory meatus, or at the base of the brain, complete deafness is nearly always present, due to implication of the auditory nerve. When the auditory nerve is spared, and the sixth and facial nerves alone affected, the lesion is almost certainly at the knee of the facial, where it bends round the sixth nucleus. If the orbicularis oris is quite free, and the remainder of the face paralysed, the lesion is in the pons; and where the orbicularis oris alone is affected, the lesion is near the hypoglossal nucleus, the tongue being also involved (Gowers).

Prognosis.—The prognosis in facial paralysis involves the question both of the possibility of recovery and the probable duration of the condition. The possibility of recovery is determined by the nature of the lesion; the probable duration of the affection can usually with fair accuracy be determined by the electrical reactions of the muscles on the paralysed side during the first two weeks.

If the lesion be in or above the nucleus, there is little chance of recovery when it is produced by sclerosis, tumour, or anterior polio-myelitis, but cases due to hæmorrhage or syphilis may recover. When the lesion is infranuclear, the prognosis is usually favourable in cases of infective neuritis, or in middle ear disease if the condition is amenable to treatment; but in basal meningitis, aneurysm, or tumour, or when caries of the petrous bone is present, the prognosis is very bad. The appearance of contractures, according to some authors, is unfavourable. As to the time of recovery, the cases may be divided into three great classes according to the condition of their electrical reactions, namely, slight, medium (Erb's "Mittelform"), and severe.

In slight cases the electrical reactions of the muscles are unaltered, except for a slight increase in the irritability during the first two or three days; recovery is usually perfect, and takes place within three weeks.

In the middle form there is only a slight increase of irritability for the first few days, but about the end of the first week there is a distinct diminution of reaction to the faradic current. This does not progress far, and is not usually great in amount. The reaction of degeneration appears in the muscles in the second or third week, and may be very complete. The muscles, when stimulated indirectly through their nerve, react normally, but when stimulated directly the reaction is abnormal. The contraction is slow, and A.C.C. preponderates. The prognosis in these cases is fairly good, recovery usually taking place in four to eight weeks, is sometimes delayed for ten weeks, and is very perfect. Mobility is often recovered before the electrical reactions are normal.

In the third class of cases, the severe form, the reaction of degeneration sets in rapidly and completely. The irritability of the branches of the nerve is quickly lost, and the muscles are characteristically altered as regards their response to faradic, galvanic, or mechanical stimuli. The prognosis in these cases is very unfavourable. Recovery may take place within three or four months, but frequently not for twelve or sixteen. It may not be quite complete, and usually supervenes after a period of stiffness and contracture which leaves permanent traces.

Treatment.—When the cause of the paralysis is known, the treatment should be directed to its removal. If syphilitic, potassium iodide and mercury must be used as in any other syphilitic case, and when there is disease of the middle ear, free exit should be provided for any discharge, and the parts kept as aseptic as possible. Rheumatic cases should be treated by free diaphoresis and a purge in the early stages, followed by continuous small doses of mercury until the active inflammation is past. The diet should be regulated, and an alkaline diuretic mixture given. Hot fomentations may be applied to the ear in the early stages, and later a blister on the mastoid process—or on the occiput if the lesion is basal—may be of some avail. At this stage, tonics such as quinine and strychnine are useful. As far as can be ascertained, electricity in no way checks the actual degeneration of the nerve, but it is certainly of use in exciting the muscles to movement, and thus keeping up their nutrition. It is doubtful whether faradism is of much effect, but the voltaic current is undoubtedly beneficial, since it stimulates both nerve and muscle. The current used should be the weakest that will produce contraction, and the application should be made daily for fifteen minutes. The positive electrode is placed below the mastoid, and the negative electrode stroked along each of the facial muscles in turn.

Little or nothing can be done for the contracture, but gentle massage along the line of the muscles may do good. Faradism is, if anything, harmful, but a weak continuous voltaic current is said to improve the condition.

FACIAL SPASM.

Spasm of the muscles of the face (mimic spasm, *tic convulsif*) may occur primarily, or it may be secondary to paralysis of the facial nerve. Primarily, it may appear in the course of a convulsive disease, such as chorea, hysteria, athetosis, tetany, or epilepsy. It may affect one or both sides of the face, or select certain muscles only on one or both sides, the orbicularis palpebrarum and the zygomatici being those most commonly involved. The spasm may be clonic or tonic, or both forms may be present. Most commonly it is purely clonic, and it is rare to find the tonic variety alone. Isolated spasm of the orbicularis palpebrarum (blepharospasm) will be described independently of facial spasm.

Etiology and pathology.—Facial spasm may occur as an idiopathic affection, or it may be due to organic lesion in the course of the facial motor tract. The idiopathic form is almost unknown in youth. It has rarely been known to occur before 20, and the most common age for its appearance is between 45 and 60. The only form that occurs in childhood is the facial spasm which succeeds facial paralysis. It is said that women are more subject to spasm of the facial muscles than men, but this is doubtful.

A distinct heredity is rare, but there is one case on record in which twenty-six members of a family were affected in five generations. It is more common to find it occurring in a neurotic individual, or in a family in which there is hereditary insanity or epilepsy. The exciting cause may be some general physical depression, such as anæmia or neurasthenia, but more frequently the spasm is brought on by some mental or emotional disturbance, such as fright, shock, anxiety, or, most commonly, prolonged worry or grief. It may occur at the climacteric, or in connection with pregnancy or the puerperal period.

The spasm may be reflex, due to some morbid condition in the eye, the nasal cavities, the mouth, teeth, or skin. In such cases it may cease at once if the source of irritation be removed, but frequently it persists. More remote irritation has also been shown to produce facial spasm, such as malposition of the uterus, or morbid conditions in the intestine, the presence of worms more especially. It has been known to occur in patients suffering from migraine, and in rare cases has followed neuralgia of the neck. Exposure of the head and neck to cold may suffice to bring it on, the attack generally beginning with pain in the face. In cases of torticollis the spasm has been known to extend from the neck to the face. The organic lesions which produce facial spasm may be situated on the nerve itself, in any part of the motor tract within the pons and cerebrum, or in the motor centre for the facial muscles, *i.e.* the lower portion of the ascending frontal convolution.

Outside the stylo-mastoid foramen a tumour or inflamed gland may irritate the nerve, or the nerve itself may be inflamed. It is doubtful whether middle ear disease or disease of the petrous bone produces facial spasm. Gowers thinks that, in such cases, spasm, if present at all, is only transient, and is succeeded by paralysis.

The nerve may be injured in its intracranial course by tumours, hæmorrhage, meningitis, aneurysm at the base of the brain, or atheroma of the neighbouring vessels. Within the pons, tumour, softening, hæmorrhage, or abscess may produce a spasm which is usually succeeded by paralysis. Tumour of the cerebellum by pressing on the pons may produce the same effect. A limited, stationary lesion, such as softening, inflammation, or abscess in the lower part of the ascending frontal convolution, may produce persistent spasm. A fall or blow on the head or compression of the fœtal head at birth may act similarly. There is one case on record in which reflex facial spasm was produced by a tumour in the intracranial portion of the first part of the fifth nerve. The fibrillary tremors which appear after paralysis do not come under consideration here.

Symptoms.—Clonic spasm.—The only symptom of facial spasm is the spasm itself. This is usually clonic, begins in one area, and extends to the neighbouring muscles. The most common form is that in which the orbicularis palpebrarum and zygomatici are involved. In such cases, when the spasm comes on the eyes are tightly shut, the forehead wrinkled, the nose screwed up, and the upper lip and angle of the mouth pulled strongly up and out. The grimace resulting from electrical stimulation of the seventh nerve in a healthy subject is precisely similar to the appearance presented during facial spasm. In old-standing cases there is persistent slight deviation of the nose and mouth to the contracted side during the intervals between the spasms.

The contractions may be frequent and momentary like those produced by the faradic current, or single contractions may occur at intervals varying from a few seconds to some minutes. There may be brief paroxysmal attacks, both tonic and clonic, or the paroxysms may last several minutes, consisting of rapidly succeeding contractions with imperfect relaxation between. In some cases there is a period in the paroxysms during which there is complete relaxation of all the muscles except the orbicularis palpebrarum. As a rule, the contractions cease during sleep, but some cases have been described in which they persisted.

The muscles involved are, as already said, most commonly the orbicularis palpebrarum and the zygomatici. The corrugator supercilii is sometimes affected, but rarely the frontalis. In some cases the corrugator supercilii and orbicularis palpebrarum are affected on both sides when the spasm is otherwise unilateral.

Some of the muscles of the lower face (depressor anguli oris, levator menti, digastric, and platysma) are occasionally involved, but the orbicularis oris is almost never affected. Bernhard records one case in which these muscles were in a state of incessant spasm. The palatal muscles are rarely involved, but in some cases the uvula contracts at the same time as the muscles of the face. Gowers thinks that these are cases of "association," or that the seventh and eleventh nerves are affected simultaneously.

It is rare that symptoms indicating spasm of the stapedius are present, but in some cases a clicking is heard in the ear at the time of the spasm. This is well marked in blepharospasm, and even in health it is possible to produce a noise in the ears by contracting the orbicularis palpebrarum firmly. In one case in which there was continuous noise in the ears, giddiness, and right lateral deviation of the eyes, the symptoms were probably due to the creation of a negative pressure in the labyrinth and semicircular canals by the contraction of the stapedius. Taste is not affected unless the nerve has been damaged. Some abnormalities in salivation have been described, but cannot be reckoned as distinctive symptoms of paralysis of the face. There are no other vasomotor or trophic changes, and the muscles do not, as a rule, hypertrophy, although Gowers records one case in which there was hypertrophy of the platysma.

The electrical reactions are usually normal, but a few cases have been described in which the excitability was increased, and in post-paralytic cases the reactions are greatly altered.

Pain is very seldom present unless there is some complication, and sensibility is rarely impaired. Occasionally there is pain in the branches of the fifth nerve at the onset of the spasm, and in a few cases "tender points" have been found on the same nerve at the points where its various branches emerge from their foramina. It is more common to find that pressure at certain points—the foramina, the cervical spines or the wrists—arrests or lessens the spasm. The spasm of the face occasionally extends to other parts, to the muscles of mastication, the neck, or the arm, but never to the orbital muscles. In some paralytic cases it appears on the sound side of the face.

Tonic spasm may be primary or secondary. It is sometimes associated with trismus, and is present in the bilateral form in paralysis agitans, tetany, and tetanus. It is often seen secondary to paralysis and in hemiplegia, when it most frequently affects the zygomatici. In a case of unilateral tonic spasm, the muscles are rigid and immobile. There are deep persistent furrows, the mouth is drawn down, and the nose, lips, and chin deviate to the affected side. The eyebrow is raised, the palpebral fissure narrow, but the eye cannot be completely closed. There is a feeling of tension in the face, and speaking and chewing are difficult.

In cases of hysterical catalepsy, a tonic facial spasm is sometimes present. There is also an hysterical facial spasm, in which, however, the tongue is also convulsed, and the locality of the spasm varies from time to time.

The course of the disease tends to be slowly progressive, and finally stationary. It is very irregular, the patient being sometimes free from it for months at a time. It may persist for years, and continues very often throughout the rest of life.

Blepharospasm is the bilateral, partial facial spasm in which the orbicularis palpebrarum is contracted on both sides. It may be tonic or clonic in nature. The tonic form is usually due to the photophobia induced by inflammation in the eyes, but may be due also to disease of the fifth nerve. The spasm in those cases may continue after the cause has been removed, and may be followed by temporary blindness from disease of the eye. The clonic form is known as nictitation, and is seen in hysteria, or as habit-chorea in nervous children.

Diagnosis.—The diagnosis is very easy, the only point of any difficulty being the distinction between pure facial spasm and that which occurs secondarily to paralysis or as a part of a general convulsive disease. In the post-paralytic form it will be found that the muscles are weak and atrophied, that there is a tendency to over-action and contracture, and that the electrical reactions are abnormal. When the face is affected in chorea, the history and the presence of convulsive movements in the limbs will give the clue; and similarly in hysteria, the history, the nature of the spasm, which is tonic, or quick and quivering, unlike true facial spasm, will determine the diagnosis.

The seat of the lesion is determined by the nature and extent of the spasm. If it is partial, and limited to the zygomatici, the injury is probably cortical; and if the muscles show increasing weakness, it is certainly an organic lesion. Tender points in the fifth nerve, or the presence of pain, indicate that the spasm is reflex, and due to some irritation of the fifth nerve.

Prognosis.—The prognosis as to recovery is unfavourable unless the spasm is reflex, and due to a remediable cause. Even in such cases it may persist after the irritation has ceased, and many cases which have apparently been cured show remissions after a time. If a case has persisted for more than a few months, its ultimate recovery is very doubtful, except in the case of blepharospasm, which may be arrested even after it has lasted for months. Although the prognosis as to life is favourable, except in cases of grave organic lesion, the annoyance caused by the disfiguring spasm may lead to intense mental depression. When the spasm occurs in the course of chorea or hysteria, the prognosis depends upon the extent of the general disease.

Treatment.—When the spasm is due to organic lesion, the treatment varies according to the nature of the disease. When due to cold, hot applications should be made to the face, and free diaphoresis induced. Any reflex irritation—a carious tooth, or morbid condition in the nose or eyes—should be removed. In all cases attention should be paid to the general health—fresh air, sunlight, good and abundant food, being of the utmost importance. Iron and arsenic should be given in anæmic cases, and hypodermic injections of strychnine are of use as a tonic.

To quiet the spasm, the bromides, in doses of 30–40 grs. daily, are useful, but there is no doubt that morphine, injected subcutaneously, either alone or combined with atropine, is the most efficacious drug. Unfortunately the injections must be kept up for many months to do any good, and the risk of forming a morphine habit is very great. Other sedatives—cannabis indica, curara, gelsemium, amyl nitrite—have all been tried without much effect. Counter-irritation to the nape of the neck or the mastoid process may do some service.

The results of the use of electricity are unsatisfactory. Surgical treatment has not hitherto given good results. The nerve may be stretched or divided; but when stretched sufficiently to allay the spasm, paralysis of the face follows, and when stretched slightly, the relief obtained is not great, and only temporary. Section of the nerve arrests the spasm, but causes facial paralysis—a condition often preferred to the spasm by the patient.

Subcutaneous section of one of the branches of the fifth nerve has been tried in cases where pressure on one of the foramina gave relief.

The treatment is to remove the cause of the spasm. Cocaine may be instilled into the eye, or cold douches applied. Sedatives, such as the bromides and belladonna, may be used, and when indicated, iron and quinine may be given as tonics. Persistent blepharospasm and nictitation in children may be cured by immersion of the face in cold water.

THE FIFTH NERVE.

PARALYSIS.

Etiology.—The lesions which affect the fifth nerve may be either intracranial or extracranial. Secondary neuritis is the most common extracranial lesion. Primary neuritis due to cold, gout, or syphilis is comparatively rare. The form of neuritis which produces herpes zoster very frequently affects the fifth nerve. Some of the branches of the nerve may be injured by stabs or bullet wounds in the mouth, or they may be involved in the growth of a tumour of the parotid gland or in the spheno-maxillary fossa. The first division of the nerve, the ophthalmic division, may be affected by growths in the pituitary body or aneurysm of the internal carotid, and it may also be involved in tumours or cellulitis within the orbit.

At the base of the brain the nerve may be injured by tumours, chronic meningitis, by syphilis, or caries of the bones, and within the pons it may be involved in tumours, hæmorrhage, softening, sclerosis, and the chronic diseases such as bulbar paralysis, tabes, and syringo-myelia. The sensory nucleus is rarely completely involved, its extent is so great; while even in widespread disease of the medulla and pons the motor nucleus may escape. The descending root is said to be sometimes degenerated in facial hemiatrophy (Mendel).

It is doubtful whether supranuclear lesions affect the nerve as a whole, and there is only one case recorded in which a supranuclear lesion affected the motor fibres. This was a case in which the muscles of mastication were paralysed by a small focus of softening in the lower third of the left anterior central convolutions.

Symptoms.—The symptoms of interference with the fifth nerve may be merely sensory, or both sensory and motor, according as the nerve is partially or wholly paralysed. The sensory symptoms may be ushered in by pain of a sharp, darting, or burning character, or by a feeling of icy coldness, with tender points at the bony foramina. There is increased sensitiveness in the area supplied by the nerve, and sometimes there are twitchings in the facial muscles, or in the muscles of mastication. The duration of this period is variable, and in many cases it does not appear at all.

The mucous membranes and the skin in the affected areas lose their sensibility, first to pain, and then to touch. The lips, the mouth, and the anterior two-thirds of the tongue become quite insensitive as far as the middle line, and in some cases the back of the tongue and the anterior surface of the palatal arch are also insensitive. In other cases only the anterior two-thirds of the tongue become anæsthetic. In consequence of the anæsthesia of the lips, mouth, and tongue, the cup used in drinking may seem to have a piece out of it on the affected side, and the food taken into the mouth is not properly chewed, because the mucous membrane has lost its sensibility and the muscles are imperfectly co-ordinated and sometimes weak. The absence of the friction of the food on the insensitive side allows that half of the tongue to become more deeply coated with fur than is the healthy side. The reflexes from the nose and eyes may be abolished, so that ammonia or snuff has no effect on the nasal mucous membrane, and a threatened injury to the cornea does not produce the usual reaction. The sense of smell is impaired owing to the dry condition of the nasal mucous membrane.

The condition of the sense of taste varies greatly in individual cases, so that no hard and fast rule can be laid down. As a rule it is lost on one-half of the tongue, and sometimes it is completely destroyed, but as the exact course of the nerves supplying the sense of taste is not yet clearly understood, it is not possible to say why this should be (see p. 539). The only point at which a lesion produces an unvarying result is on the lingual nerve after it has been joined by the chorda tympani. In such cases the loss of taste is limited to the anterior two-thirds of the tongue on the affected side. Trophic changes are present in varying degrees. The face may be paler or redder than normal, or its vascularity may be quite unaffected. The hair on the affected side may become greyer and thinner, and the secretions may be increased or diminished according as irritation or paralysis is present. The tears are sometimes suppressed. Injuries to the cheek do not heal readily, and there is a tendency to ulceration or hæmorrhage in the lips, gums, and inside of the cheeks, probably due to unconscious self-injury by biting the insensitive parts. The teeth may become loose. When the injury is at the base, the eyeball may inflame, producing the condition commonly, though wrongly, termed neuroparalytic ophthalmia, in which the cornea becomes opaque and ulcerated, and destructive ophthalmitis sets in. The whole eye may become purulent; the infection may spread to the cerebral membranes and cause death by secondary meningitis. This condition is usually present when the lesion is in the Gasserian ganglion or the distal portion of the nerve, but does not appear when the lesion is situated in the pons or higher. It is due not merely to the insensibility of the conjunctiva, but rather to an irritative inflammation of the nerve (Ferrier).

Herpes zoster occasionally appears in the area supplied by the fifth nerve, and is said to be due to softening of, or hæmorrhage into, the Gasserian ganglion. It is usually preceded and accompanied by great pain and impairment of sensibility. In old people the attack of herpes is often followed by severe and intractable pain, which may last for months or years.

The motor symptoms in affections of the fifth nerve are limited to paralysis or paresis of the muscles of mastication, namely, the masseter, temporal, external and internal pterygoid, mylo-hyoid, and digastric muscles. This is first observed by the patient himself as an increasing inability to chew hard food. The muscles are readily tired, and in long-standing cases become so weakened and atrophied that chewing may be almost impossible. The weakness of the muscles may be recognised by placing the fingers on the masseter and temporal muscles and directing the patient to bring the jaws firmly together as in biting. If the muscles are merely paretic, the contraction will be later on the affected side; but it will be entirely absent if paralysis is complete. The jaw cannot be moved to the sound side, and when depressed tends to be displaced towards the paralysed side. When the mouth is widely open, the condyle of the jaw is slightly depressed on the sound side, but is prominent on the affected side, since the paralysed temporal muscle cannot fix the condyle, while the mylo-hyoid and digastric do not pull the jaw down on the affected side. When the paralysis has existed some time the muscles atrophy, the zygoma appears abnormally prominent, and distinct hollows are present above and below it. Chewing becomes impossible in late stages. The condition of the tensor palati and tensor tympani muscles in paralysis of the motor branches of the fifth nerve is as yet not sufficiently well known to permit of a definite statement concerning them.

The electrical reactions of the muscles are considerably altered. There may be no response to either galvanic or faradic current when applied directly to the muscles. In other cases the reaction of degeneration is present when the galvanic current is applied directly. In bulbar paralysis the response is quantitatively diminished.

Diagnosis.—When there is marked paralysis in both sensory and motor branches of the fifth nerve, or when the sensory part is affected without there being anæsthesia in any other area, it is not difficult to decide that paralysis of the fifth nerve is present. The area supplied by the fifth nerve may be included in a general hemianæsthesia, but in such cases the extent of the paralysis, and sometimes the loss of some of the special senses, will indicate the nature of the condition. When the nerve is merely irritated, great pain may be present, and it may be difficult to decide whether or not it is a case of neuralgia. The condition of cutaneous sensibility in the face, and of the sense of taste, should be carefully examined. When the causal lesion is organic, the cutaneous hyperæsthesia is better marked, and persists more constantly than in neuralgia, and the pain has not the same tendency to extend to other nerves. Otherwise the symptoms may be identical.

The seat of the lesion may be determined by the area of anæsthesia. When the lesion is in the Gasserian ganglion, at the base of the brain, or in the pons, all the branches of the fifth, including the motor, are affected. If it is in the pons, the paralysis is usually bilateral; and if it is in the posterior limb of the internal capsule, there is usually hemianæsthesia, a condition which is very difficult to distinguish from hysterical hemianæsthesia. If the onset is sudden, the lesion is probably acute and grave, most commonly hæmorrhage or softening; but when slow, a chronic disease, such as tabes, syringo-myelia, or acute or chronic disease of the cord, may be suspected. In such cases trophic changes are usually well marked.

Bilateral paralysis, when other nerves are implicated, is due to bulbar paralysis or polio-encephalitis superior, whilst unilateral paralysis of all three branches, or of two branches, is characteristic of tumour of the middle fossa of the skull. Affection of the superior division of the nerve alone indicates that the lesion is in the orbit or sphenoidal fissure; and if the second division alone is affected, the spheno-maxillary fissure or the superior maxilla itself will be found to be the seat of the disease. The third division is rarely affected alone, but is usually involved along with the second.

The nerves which are most commonly paralysed along with the fifth are the third and the sixth. The seventh and eighth are not so frequently implicated. If the sixth, the third, and all three branches of the fifth are paralysed simultaneously, the lesion must be at the front or side of the pons.

Prognosis.—The prognosis in paralysis of the fifth nerve depends upon the nature and seat of the lesion. It is necessarily grave as regards life when there is a progressive intracranial disease; but when the paralysis is due to a remediable cause, such as syphilis or cold, the prognosis is much more favourable.

Treatment.—If syphilis be suspected, a course of antisyphilitic treatment should be prescribed; and if inflammation be present, a blister may be applied on the occiput or mastoid processes, but should never be used on the temple, as ulceration may be easily produced. Rheumatic cases should be treated with hot fomentations during the first few days, but not later, in case vesication occur. The pain may be treated by the use of phenacetin or antifebrin, by gelsemium, or by the external use of veratrine ointment. Local subcutaneous injections of cocaine or morphine sometimes act very quickly, or a weak voltaic current may be used if the pain is not very severe.

When anæsthesia is present, it is wiser not to use stimulating liniments, as they may easily cause trophic changes. Faradisation may improve nutrition, and restore functional activity if the nerve or centre has not been so severely injured as to produce permanent interruption. One electrode may be placed on the occiput or behind the ear, and the other, in the form of a fine wire brush, applied to the face. The current should be strong enough to leave a sensation for two or three minutes.

If the eye be anæsthetic, care should be taken to protect it from injury by foreign bodies. It should be kept covered, and the conjunctiva frequently washed with a half-saturated solution of boracic acid.

FACIAL HEMIATROPHY.

Atrophy of one-half of the face is a comparatively rare condition, the causation and pathology of which are still obscure. The ascertained facts seem to indicate that it appears in slightly different forms, from more than one cause.

Etiology.—It usually appears in childhood or early youth, but has occurred in adults, and even late in life. It is rather more common in the female sex. Heredity plays no part in its etiology. It may be without apparent exciting cause, or it may follow some local lesion or general disease. The following local injuries have been observed:—extraction of a tooth, a blow on the angle of the orbit, contusion of the upper or lower jaw, a blow on the face, exposure to cold, inflammation of the superior maxilla, neuritis of the fifth nerve, and tumour of the pons. The general diseases which have apparently given rise to the condition are scarlatina, typhoid fever, diphtheria, influenza, and acute rheumatism.

In some cases there is a congenital disparity in the size of the two halves of the face, which must be distinguished from true facial hemiatrophy, which only begins some years after birth. In the congenital cases the disparity is not so marked, nor is the condition progressive.

Pathology.—The pathology of the condition is very little known, but in most instances the fifth nerve is abnormal. In the case examined by Mendel there was a distinct interstitial neuritis, and in that described by Homén there was a parenchymatous neuritis in all three branches of the fifth nerve, the result of a tumour in the middle fossa of the skull. There is little doubt that in some instances the fifth nerve is the seat of a peripheral affection, due either to local or remote irritation, as is shown by the limitation of the atrophy in some cases to the area supplied by one division of the fifth. In other cases the lesion is probably in the Gasserian ganglion or higher, and in some others an affection of the sympathetic is the cause of the condition. Mendel and Homén both describe changes in the nuclei and roots of the fifth nerve. The cases on record are still too few to allow a distinct opinion to be formed.

Symptoms and course.—In a typical case the atrophy begins at one spot, usually on the cheek. The skin becomes thin, glazed, pale, and slightly depressed below the surface of the healthy skin. Any hairs that may be on the part become grey and thin, and finally drop out, while the sebaceous follicles waste. The atrophy spreads gradually, involving skin, subcutaneous tissue, and bones, but not the muscles, which maintain their size and activity. In an advanced case, especially one which has begun in early life, the appearance is very striking. The forehead, cheek, and lower jaw are much smaller on the affected side, the hair is grey, and the eye sunken from atrophy of the orbital tissues. The skin is thin and glazed, closely applied to the structures below, and marked with furrows and wrinkles which do not appear on the healthy side. The vertical median line is distinct, separating the atrophied from the normal half of the face; the nose is deflected to the atrophied side, and the ear may be small; the teeth are small, decayed, or may drop out altogether. The muscles, however, in nearly all cases, are quite unaffected, their electrical reactions normal, and their movements unimpaired, except in the case of the muscles of mastication, which may be atrophied, or be subject to spasm.

In addition to the trophic changes there may be some sensory disturbance. In some cases there is neuralgic pain, and in others anæsthesia, partial or complete, is present in the areas supplied by one, two, or all of the divisions of the fifth. Taste has sometimes been lost on the anterior half of the tongue on the affected side. The organs of special sense are unaffected, and the saliva and tears are normal; but in a very few cases the tongue and palate of the affected side are wasted, and in some others opacities form on the cornea. Vasomotor changes are not equally present. As a rule, the atrophy is strictly confined to the area of distribution of the fifth nerve, but one case has been described in which it was bilateral, and in Virchow's case the skin on the back and arm of the same side was much atrophied.

The disease usually begins in childhood or early youth, and advances steadily for some years, when its progress is arrested, and it remains stationary for the rest of life. It is usually uncomplicated by other disease, but one case is on record in which there were a number of exostoses on the face at the same time as the atrophy.

Diagnosis and prognosis.—There is little difficulty in diagnosing facial hemiatrophy, the only condition with which it is likely to be confounded being the congenital disparity in the two halves of the face, already described, in which, however, the difference is not great, the hairs not affected, and the changes in the skin not so marked. The prognosis is bad, except in cases which have appeared in adult life after an injury to the fifth nerve. These sometimes recover.

Treatment.—This is directed towards the cause, should that be removable, but, unfortunately, very little can be done. If any improvement takes place, it is probably due to the removal of the cause of the condition.

MASTICATORY SPASM.

Spasm of the muscles of mastication may be either *tonic* or *clonic* in character. In the tonic form the jaws are firmly closed, producing the condition known as lockjaw; the teeth cannot be separated at all, or at most for a few millimetres; the masseter and temporal muscles stand out hard and rigid, and may be painful from excessive contraction. The jaw cannot be moved passively, any attempt to do so by force causing great pain in the muscles. While the spasm lasts, eating is quite impossible. The condition is almost invariably bilateral.

Etiology and pathology.—The spasm may be due to local irritation in the distribution of the fifth nerve, *e.g.* from a carious tooth, or ulceration in the mouth, especially if the caries be in the last molar, and cause an alveolar abscess. Eruption of the last molar has been known to bring on a spasm which lasted for months. In some rare instances sensory irritation elsewhere has produced spasm of the muscles of mastication. Cold has also been known to do so in a few cases, most of which were, however, young women, and possibly hysterical subjects.

Central lesions in the pons, causing irritation of the motor root of the fifth nerve, may produce spasm, which is often permanent. It is bilateral in most cases, but may be unilateral.

Symptoms.—Tonic spasm is an important and early symptom of tetanus, and appears late in severe cases of tetany. It is also present in hysteria occasionally, either as a brief spasm lasting for fifteen minutes or so, or as a more permanent attack which persists until the next hysterical fit comes on, after which the jaws remain free. In a few rare cases the hysterical spasm was unilateral, the jaw being fixed in an oblique position.

Clonic spasm appears in two forms, either as repeated, rapid contractions, or as a single, sudden spasm occurring at fairly long intervals.

The first form, in which the lower jaw makes rapid vertical movements against the upper, so that the teeth clatter together, is well known in healthy persons, when exposed to a sudden fall of temperature, as "chattering teeth." It also occurs during the rigors of various pyrexial diseases, in general clonic spasm, such as convulsions, and in some cases of paralysis agitans. It is almost always bilateral and vertical. It seldom occurs as an isolated symptom, but instances have been described among elderly women.

The second form, the single clonic spasm, occurs in chorea, but is rare as an isolated symptom. In the latter case it comes on quite suddenly, without warning, often when the patients are eating or speaking. The jaws clench with some force, and if the tongue happens to be between the teeth it may be lacerated. It is most common in middle-aged men, and frequently occurs at night, the patient waking from sleep to find his tongue badly bitten. Bernhardt inclines to think that such cases are really epileptic. When the pterygoid muscles are also involved, there is a lateral movement of the jaw, causing the "teeth-grinding" to which some patients, especially nervous children, are subject during sleep.

Diagnosis and prognosis.—Spasm of the muscles of mastication is easily recognised, the only conditions with which it might be confused being rheumatoid or inflammatory disease of the joint, or tumour of the ramus of the jaw. In the former case other joints would be affected, and in the latter the condition is usually sufficiently obvious. The prognosis is very bad in cases of tetanus, and also when there is an organic central lesion, in which latter case the spasm is frequently permanent. In cases of isolated spasm there is usually improvement and gradual recovery after prolonged treatment. In hysteria, epilepsy, and tetany the condition improves under treatment of the general disease.

Treatment.—The treatment varies with the cause of the spasm. If due to local irritation, the carious tooth must be removed or the ulcer treated. When one of the molars is at fault, it may be necessary to give chloroform to relax the muscles sufficiently to allow the tooth to be drawn. Local applications should be used to relieve the pain, and tonics, such as iron and quinine, given to improve the general condition. When the spasm is due to cold, a hot-air or vapour bath may do good. Electricity is sometimes useful. The voltaic current should be employed, the positive pole being placed on the back of the neck and the negative on the muscle. The bromides are of value in lessening the paroxysmal and nocturnal spasm.

When the condition is a symptom of a general disease, such as tetanus or hysteria, the treatment is that of the primary affection. The hysterical spasm is often arrested by a blister behind the ramus of each jaw, and in tetany, the removal of any irritation (worms more especially) in the digestive tract will effect a cure. When the contraction persists for a length of time, it may be necessary to extract a tooth, in order to allow food to pass into the mouth, or to feed the patient by the nose-tube, or by means of nutrient enemata.

THE EIGHTH NERVE.

Etiology and pathology.—Deafness may be produced by a lesion anywhere in the tract from the auditory centre in the temporo-sphenoidal lobe of the cerebrum to the labyrinth. Within the cerebrum the tract may be affected by traumatism, hæmorrhage, softening, tumour, or abscess, and within the pons or medulla by hæmorrhage, softening, or tumour. It is not so frequently affected in this situation as are the other nerves and nuclei of the medulla. At the base of the brain it may be compressed by meningitis, especially the syphilitic form, by tumour, or by aneurysm. Meningitis and disease of the petrous bone may affect the nerve in the internal auditory meatus, and neuromata, calcareous deposits, and interstitial hæmorrhages have been known to occur in the nerve itself. It is probable that in cases of cerebral tumour a primary neuritis is present in the auditory nerve, analogous to the optic neuritis seen in such cases, but it is doubtful whether a rheumatic neuritis ever occurs. Primary atrophy of the nerve may take place in tabes, and a form of atrophy is also met with in advanced age, due probably to endarteritis and fatty degeneration. The auditory nerve may sometimes be affected during the course of the general infective or toxic diseases, such as typhoid fever, scarlatina, Bright's disease, or syphilis, and deafness has been known to come on rapidly in cases of mumps. Finally, deafness may occur with no apparent cause, or may be produced by some external influence such as cold, sunstroke, a plunge into water, or a concussion. Disease of the labyrinth may affect the nerve in two ways—it may simply hinder the passage of the vibrations to the end-organ, or it may injure the nerves themselves or their sensory terminations. Disease of the labyrinth is in most cases secondary to affections of the middle ear, the petrous bone, or the basal membranes. The primary affections are due to inflammation (simple or suppurative), congenital syphilis, senile degenerative processes, and hæmorrhage. The inflammatory affections may lead to fibrous and calcareous degeneration. Certain drugs—quinine, salicylic acid, chloroform—affect the labyrinth very markedly, causing deafness and giddiness. Congenital deafness is common, four-fifths of all deaf mutes being born deaf. The condition is hereditary, as is proved by the numbers of deaf mutes present in families in which the condition exists, and in which intermarriage takes place. Functional deafness may occur in hysteria, or in the sudden anæmia following severe hæmorrhage.

Symptoms.—The symptoms of affections of the auditory nerve may be irritative or paralytic. Irritation of the nerve gives rise to subjective noises (tinnitus), hyperæsthesia of the nerve, giddiness, vomiting, and impairment of the equilibrating power. Paralysis of the nerve produces deafness, varying in amount from slight weakness of hearing to total deafness. Both the irritative and the paralytic symptoms are present as a rule. The onset of the deafness is usually sudden, but may be gradual, and its course may be rapid or slow. The condition is very often bilateral.

The symptoms just described are common to disease of the nerve or the labyrinth, and also to some cerebral affections, so that the exact seat of the lesion cannot be deduced from them alone.

When the disease is in the labyrinth or nerve, the tuning-fork is heard less distinctly when it is applied directly to the bones of the head (usually to the frontal bone in the middle line, or to the mastoid process) than when it is held opposite the external auditory meatus—bone-transmission is less perfect than air-transmission. In such cases the sound of the tuning-fork applied to the bone is intensified when the external auditory meatus is closed, as the middle and outer ear are thus converted into a closed resonant cavity. When deafness due to disease of the labyrinth or nerve is only slight, so that ordinary sounds in the air are not well heard, it will be found that the tuning-fork is heard for a longer time by air-transmission than by bone-transmission. A watch with a clear, sharp tick may be used in a similar way to test the hearing. High-pitched, short sounds are not well heard in cases of nerve deafness, but low notes are easily distinguished. This can be demonstrated by the use of Galton's whistle, from which a note of any pitch can be obtained. If the high notes are heard on one side and not on the other, there is probably disease of the labyrinth or nerve on the first side; but if the notes of the whistle are not heard at all, there is probably extensive double disease.

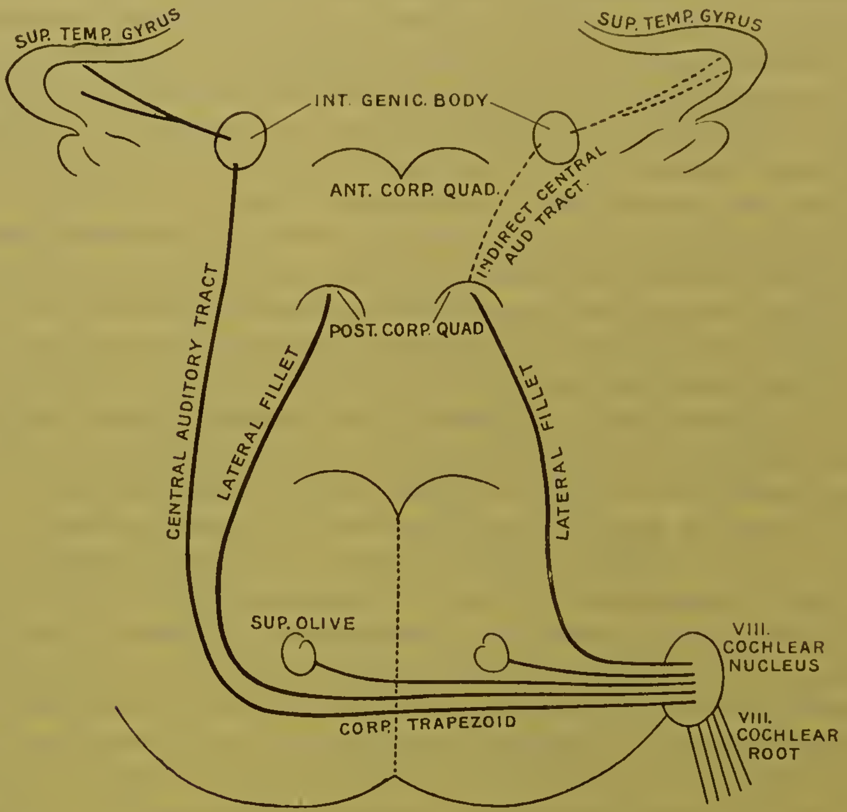


FIG. 11.—Diagram of the central path of the auditory nerve (cochlear root), showing its termination in both superior temporal convolutions.—After Ferrier and Turner.

It must be carefully borne in mind that ankylosis of the stapes or senile degeneration may cause deafness without producing any pathological change in the membrana tympani or the Eustachian tube, and thus might easily be mistaken for extensive labyrinthine or nerve disease. It has been shown that the electrical reactions are altered in disease of the nerve, but they are so difficult to obtain, and the attempt causes the patient so much discomfort, that they are of little practical value.

Diagnosis.—The diagnosis of disease of the auditory nerve is not always easy. The use of the otoscope and tuning-fork will in most cases eliminate middle ear disease, including the cases of ankylosis of the stapes, which, when superficially examined, might lead to a diagnosis of labyrinthine or nerve lesion. The symptoms which occur in disease of the labyrinth, the nerve, the pons, or cerebrum, may be exactly similar. The presence of associated paralysis is of great use in determining the seat of the lesion. Should the facial muscles be paralysed, and no middle ear or bone disease be present, the lesion is almost certainly in the internal auditory meatus or at the base of the brain, whereas if the abducens alone is involved, the lesion must be at the base of the brain. Sudden deafness accompanied by paralysis on the other side of the body points to a lesion in the pons or medulla, but when unaccompanied by paralysis elsewhere, would suggest hæmorrhage into the nerve or labyrinth, or embolus of the auditory artery.

Bilateral deafness may be due to chronic degeneration of the labyrinth, or to acute inflammation. It is, of course, sometimes secondary to double otitis. It may also be due to disease of the medulla, involving both auditory nuclei, or to tumours of the corpora quadrigemina, or, lastly, to symmetrical lesions in both temporo-sphenoidal lobes, as occurred in one case of syphilitic gumma.

It is sometimes impossible to distinguish between disease of the labyrinth and disease of the nerve. It has been observed that in nerve deafness the higher tones go first, and that the duration of perception of the C_2 tuning-fork (Schwabach's test) is greatly shortened. Disease of the labyrinth cannot be diagnosed unless it is possible to exclude a lesion of the middle ear on the one hand, and of the nerve and its higher centres on the other.

Treatment.—In the great majority of cases the only treatment of disease of the auditory nerve is the treatment of the cause when possible. Counter-irritation is of great use sometimes, especially in acute cases. It may be applied in the form of blisters over the mastoid. Electricity has proved of little or no value in the treatment of the auditory nerve.

THE NINTH NERVE.

Paralysis.

Etiology and pathology.—The glosso-pharyngeal nerve may be affected by a lesion within the medulla at the base of the brain, or high in the neck. The lesions in the medulla, which may injure the emerging nerve itself or its nucleus, are acute bulbar paralysis, due to embolism, thrombosis, or hæmorrhage, or chronic bulbar paralysis, in which case the cells of the motor nucleus are involved. The roots of the nerve may be implicated in aneurysm of the vertebral artery, tumour, or disease of the membranes or bones at the base of the brain, and in injuries to the base of the skull. The nerve is so high in the neck and so deeply situated that it is seldom implicated in operations or wounds in that region.

Symptoms.—The nucleus and trunk of the glosso-pharyngeal nerve are so closely associated with the other nerves rising from the medulla, that it is never injured alone, and therefore the symptoms which its paralysis produces cannot be ascertained definitely. The anaesthesia described in some cases as present in the gums, soft palate, and the upper surface of the epiglottis is probably due to paralysis of the fifth nerve, and the difficulty of swallowing is due to the implication of the hypoglossal, the spinal accessory, and the vagus. It is generally supposed that paralysis of the glosso-pharyngeal would cause loss of taste on the posterior part of the tongue, but even on this point evidence is not at all conclusive. The fibres of the glosso-pharyngeal have been proved experimentally to end in the circumvallate papillæ at the posterior part of the tongue; but no case has been recorded in which an injury to the roots of the nerve has impaired taste in this part, except one recorded by Pope, in which an aneurysm of the vertebral artery compressed the roots of the left glosso-pharyngeal nerve, caused complete loss of taste in the left half of the tongue posteriorly, and incomplete loss in the anterior half. On the other hand, there are cases on record in which the glosso-pharyngeal was paralysed and taste was unimpaired, and also cases in which paralysis of the fifth nerve produced loss of taste in both the anterior and posterior parts of the tongue. It has been observed that in some cases of middle ear disease the sense of taste was lost at the back of the tongue, the tympanic plexus, which includes Jacobson's nerve, having been injured by the disease. It is possible, of course, that in these cases the chorda tympani was also injured. Gowers supposes that the taste fibres from the back of the tongue must leave the ninth nerve at its petrosal ganglion, then pass by Jacobson's nerve through the tympanic plexus, and thence through the small superficial petrosal and the otic ganglion to the third division of the fifth nerve.

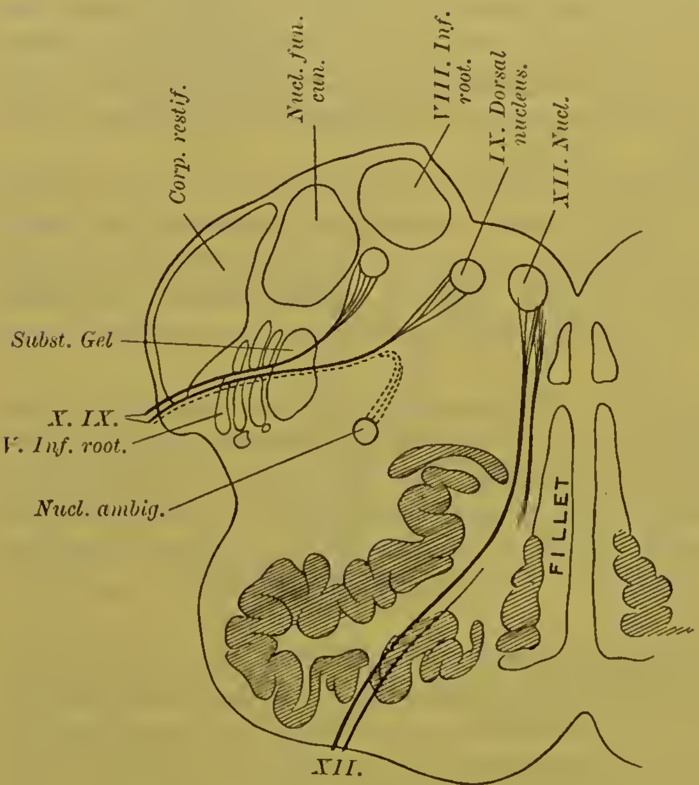


FIG. 12.—Diagram to show the mode of origin of the hypoglossal and vago-glosso-pharyngeal nerves from the medulla.

Anaesthesia of the pharynx is produced by a lesion of the glosso-pharyngeal nerve, and may contribute to the difficulty of swallowing. This anaesthesia also extends to the upper part of the pharynx, the Eustachian tube, and the tympanic cavity, which are supplied with common sensation by the glosso-pharyngeal nerve. The movements of the œsophagus may be slightly impaired by paralysis of this nerve, since it has been shown that, after section of the nerve, the muscles of the œsophagus remain in a state of tonic contraction for some time. When the tympanic plexus is injured in middle ear disease, there may be a disturbance of the secretion from the parotid gland.

Prognosis.—The prognosis depends upon the nature of the lesion. When the paralysis occurs, as it nearly always does, in association with paralysis of the neighbouring nerves in chronic or acute bulbar disease, the prognosis is very grave. It is better, however, when syphilis, middle ear disease, or the functional neuroses are the cause of the condition.

Treatment.—Very little can be done for paralysis due to a lesion in the medulla. Electricity may be employed, and a course of iodide of potassium may benefit the patient temporarily. Antisyphilitic remedies are to be employed when specially indicated, and the local condition must be treated when the middle ear is diseased. Hysterical and hypochondriacal paralysis will yield to the general treatment of these conditions.

THE TENTH NERVE.

Paralysis.

Etiology and pathology.—Paralysis of the vagus may be due to affections of the nucleus or of the nerve trunk. The nuclear lesions are softening, hæmorrhage, tumour, or degeneration caused by tabes, multiple sclerosis, bulbar paralysis, or syringomyelia. The lesions which affect the nerve may occur near the roots or in the further course. Near the roots we find meningitis, especially the syphilitic variety; growths from the meninges or bones; caries (syphilitic more especially) of the bones; and aneurysm of the vertebral artery.

In the neck, the nerve may be injured by punctured or gunshot wounds, or during operations, such as the removal of glands, of tumours of the neck, or an enlarged thyroid gland, or ligature of the carotid artery. Pressure from tumours or glands is most likely to occur near the base of the skull or at the upper part of the thorax. Within the thorax, aneurysm of the great vessels or enlargement of the mediastinal glands may compress the nerve, especially its recurrent laryngeal branches, or it may be included in pleuritic or pericarditic thickenings or in malignant growths in the mediastinum. Toxic or infective neuritis may cause paralysis. The neuritis may be due to alcoholism or lead-poisoning, or may appear as a complication or sequel of an acute infective disease, most commonly after diphtheria, typhoid fever, and septicæmia. Neuromata in the course of the nerve may impair its function.

Symptoms.—The course and distribution of the vagus are so extensive, and its functions so numerous and important, that the symptoms produced by a lesion affecting it are necessarily very variable and very complex. When, in addition, we consider the frequency with which other nerves are implicated in the same lesion, it will be seen that the symptomatology of affections of the vagus may become exceedingly involved. The symptoms may be classified according to the part of the vagus affected; the main trunk, or the laryngeal, pharyngeal, pulmonary, cardiac, œsophageal, or gastric branches.

Laryngeal branches.—The fibres of the vagus which supply the larynx are all derived from the internal branch of the accessory nerve; they pass to the larynx as the superior and inferior (or recurrent) laryngeal nerves. In bilateral paralysis of both these nerves, the vocal cords assume the cadaveric position, they become slightly and equally separated from each other. There is no movement on inspiration or attempted phonation, the voice and the power of explosive cough are completely lost, and there may be stridor on inspiration if the current of air approximates the cords. This condition is not common, and is only found in central lesions where the nuclei are affected. When one cord only is paralysed, it remains motionless in a position of slight abduction, and may become concave at its edge, thin and atrophied. The other cord acts normally on inspiration and phonation, or it may even cross the middle line. The voice in this case is hoarse, feeble, and low-pitched. There is no stridor, and in cases where the paralysis is slight the power of explosive cough may be preserved.

In many cases the paralysis of the muscles of the larynx is partial, some of them being left unaffected. The chief abductors of the larynx, the crico-arytenoidei postici, are of all the laryngeal muscles the earliest and most severely affected. When they are paralysed on both sides, the cords are approximated, and the power of abduction is lost almost entirely. During phonation or coughing they approach each other more closely, but the normal recession during inspiration does not take place. In a case which has lasted some time the glottis may become further narrowed by contracture of the adductors. The voice is not affected, since the cords are in the position for phonation, and coughing takes place normally. On inspiration, not only do the cords not separate, but there is actual approximation by the current of entering air, and consequently inspiration is lengthened and loud stridor produced. There is no expiratory stridor. The condition is a serious one, as is shown by the appearance of the patient. The larynx works violently up and down; there is urgent dyspnœa, the face is livid, and the lips cold. A slight catarrh of the larynx may at any moment entirely close the glottis, and immediate tracheotomy be necessary. The absence of expiratory stridor, and the preservation of the voice, distinguish this condition from laryngeal spasm.

When the abductor is paralysed on one side only, the opposite one continues to act, and the symptoms are not at all marked as a rule, dyspnœa and stridor being absent, or only very slight.

When the adductors of the larynx are paralysed, the cords cannot be approximated beyond the cadaveric position in phonation, but can be fully abducted during inspiration. There is no stridor, the voice is lost, but the patient can cough, and in cases of hysterical aphonia may be able to sing.

Pharyngeal branches.—The pharyngeal branches of the vagus reach the pharynx by way of the pharyngeal plexus. Their paralysis gives rise to great difficulty in swallowing, as the food is not properly grasped by the constrictors. Pulpy food is more easily swallowed than either fluids or solids. When the superior constrictor alone is paralysed, the action of the middle sphincter is said to force the food into the nose. This might occur if the palate were also paralysed, but is not likely to happen as long as the palate is unaffected. Choking and spasm of the larynx from irritation by particles of food are very common. In unilateral paralysis of the pharynx, the difficulty in swallowing is much less. In two such cases, Erb describes a dilatation of the pharynx, posteriorly and externally, on the paralysed side; but in another case the affected half of the pharynx was contracted, and showed a bulging forward.

Pulmonary branches.—The pulmonary branches of the vagus contain both efferent and afferent fibres, and join with branches from the inferior cervical and great dorsal sympathetic ganglia and the cardiac plexus to form the pulmonary plexus. Experiments on animals have shown that these fibres are of two kinds, regulating and inhibitory.

In man, injury to or section of one vagus does not appear to produce marked effects on respiration; but there are some cases on record in which section of the vagus produced slow, deep snoring, laboured breathing, laryngismus, hoarseness or complete loss of voice, changes in breath-sounds on the affected side, dyspnœa, threatening suffocation, and small rapid pulse. The pneumonic patches in the lung seen so frequently after injury to the vagus, are thought by many to be due merely to the presence of particles of food which have passed through the paralysed larynx. This is the more likely, as the congestion or œdema are not invariable. In some cases of tachycardia associated with emphysema, the condition is probably the result of some affection (neuritis (?)) of the vagus which caused contraction of the bronchial muscles. Hiccough, which is produced by clonic spasm of the diaphragm, is undoubtedly originated in many cases by some disorder of the respiratory centre; and the same may be said of many cases of periodic asthma.

Cardiac branches.—The fibres which pass from the vagus to the cardiac plexus exert a continuous, regulating, and an inhibitory influence on the heart, as is proved by the fact that paralysis of the vagus greatly accelerates the heart's action, whereas stimulation of the nerve greatly reduces its rate. It has been possible in a few cases to stimulate the nerve by pressing directly upon it in the neck, or by compressing a tumour in which the nerve was involved. The heart can thus be slowed, or even arrested for one or two beats. In some toxic conditions also, gout especially, the vagus is stimulated, and the heart-beats slowed.

The acceleration of the heart may be due to atrophy of the vagus from compression by a tumour or enlarged glands, or it may be brought on by inclusion of the nerve in a ligature during operation, or it may be due to a parenchymatous neuritis occurring as sequel to an acute infective disease, such as diphtheria, typhoid, or influenza. In these cases the heart may beat from 90 to 150 or even 200 in the minute, and the pulse is soft and full.

Angina pectoris has occurred in some cases in which the branches of the vagus were involved in disease of the cardiac plexuses; and one case of neuritis of one vagus is mentioned in which pseudo-anginal attacks were present. Experiments on birds have proved that the vagus contains trophic fibres for the heart, and in man also the cardiac muscle has shown fatty degeneration after disease of the vagus.

Œsophageal branches.—Paralysis of the œsophageal branches of the vagus may cause some difficulty in swallowing, and give rise to symptoms resembling those of stricture. The passage of a bougie serves to distinguish the conditions. Spasm of the œsophagus occurs much more frequently.

Gastric branches.—Since the vagus is the sensory, and to some extent also the motor nerve of the stomach, it follows that disease in its course may produce marked gastric symptoms. The appetite may be greatly impaired or lost, but may be distinctly increased, and in some cases is insatiable. It may be quite normal. Vomiting may occur, either from a central cause such as meningitis, from interference with the nerve in its course, or with its end-branches in the stomach, or, lastly, from irritation of the branch supplying the external auditory meatus. In some cases there is intense neuralgic pain in the stomach. This is especially common in hysterical patients, and is accompanied by other symptoms of a vagus neurosis, such as vomiting, abnormal pulse, and lowered sensibility in the larynx and pharynx. The pain is in such cases independent of meals.

The gastric crises of tabes are probably due to a lesion in the vagus, either in the nerve or its nucleus in the medulla.

Diagnosis.—The diagnosis of paralysis of the vagus may be very easy or it may be extremely difficult, according to the seat, extent, and character of the lesion, and the condition of the neighbouring nerves and nuclei. The points which guide us in forming a diagnosis are the conditions present in the larynx, the lungs, the heart, and the stomach, which should all be carefully examined. When an injury or operation in the neck is followed by laryngeal paralysis, or by some of the other symptoms already described as dependent upon injury to the vagus, the diagnosis is easy; but there are other cases in which the lesion is not so evident, and the paralyses are variable and atypical, which require careful investigation.

The neck and mediastinum should be examined for tumours or enlarged glands, or for increase in the size of the thyroid gland. Failing these, it may be possible to obtain a history of recent diphtheria, influenza, or typhoid fever, and evidences of alcoholism, syphilis, or lead-poisoning should be sought for. If the disease be intracranial, some of the other nerves rising from the medulla are usually affected, and the chronic nervous diseases affecting the medulla, bulbar paralysis, lateral sclerosis, or tabes, show characteristic symptoms which would serve to distinguish them. If there is no evidence of a gross lesion anywhere throughout the course of the nerve, the paralysis will probably be due to hysteria, neurasthenia, or hypochondriasis, or may be reflex from irritation of the digestive tract.

Owing to the very long course of the branches of the vagus, it is more common for one or more of them to be affected than for the trunk to be involved as a whole. Laryngeal paralysis, especially the unilateral form, due to lesions affecting the recurrent laryngeal branch of the vagus, is the paralysis which occurs most commonly alone, but the gastric, pulmonary, or cardiac branches may be affected separately, or the roots of the nerve within the cranium.

In cases of laryngeal paralysis, there are usually signs of intra-thoracic disease; and when the roots are affected, some of the neighbouring nerves are almost invariably implicated. The position of the lesion is thus marked out by the symptoms produced. If one branch only is paralysed—the recurrent laryngeal, for instance—the paralysis will be limited to the area it supplies. If the whole trunk is affected high in the neck, it is shown by the presence of abnormalities in the rate of the heart and respiration; and probably vomiting is also present in addition to the laryngeal paralysis. When the roots are involved in a lesion, the hypoglossal and glosso-pharyngeal nerves are usually implicated also. When the lesion is bilateral, it is as a rule within the medulla, affecting both nuclei.

Prognosis.—The prognosis in paralysis of the vagus depends upon the character of the lesion. It is very grave when the condition occurs as extension of a chronic disease of the medulla, such as bulbar paralysis or tabes; or when the nerve is pressed upon by tumour in the neck, or by aneurysm, tumours, enlarged glands, or pleural or pericardial effusion within the thorax. It is also grave when the paralysis occurs during or as a sequel to an acute infective disease. On the other hand, the prognosis is fairly good when the affection of the vagus is part of a functional disease, or is due to syphilis, or to a tuberculous disease of glands which is amenable to treatment, medical or surgical.

As already said, atrophy or section of one vagus may produce little or no effect in man, but still it must be regarded as a grave condition. In laryngeal paralysis, the prognosis is grave when the abductors of the larynx (the crico-arytenoidei postici) are affected, as there is great danger of suffocation.

Treatment.—Since paralysis of the vagus is always secondary to some other disease or lesion, its treatment must be the treatment of its cause. Therefore, in syphilitic cases, antisyphilitic remedies must be employed, and when tuberculous glands are the cause of the paralysis, general treatment with cod-liver oil, iodide of iron is indicated, or, when necessary, excision of the diseased glands. The treatment of the functional affection of the nerve will be discussed in the articles on Hysteria, etc. Polyneuritis due to alcoholism or to acute infective disease is best treated by stimulating drugs, subcutaneous injections of camphor or strychnine being most useful. In the infective diseases, wine, or alcohol in any form, is often of great use. Where the paralysis is evidently a reflex neurosis, attention should be paid to the digestive tract and uterus, and any abnormalities there corrected if possible. If intestinal worms are present, a vermifuge or vermicide should be used. Any malposition of the uterus should be rectified, and if a floating kidney be the irritating agent, it should be secured in some way. Chronic diseases of the medulla must be treated generally as indicated under the different diseases.

The paralyses of the larynx, if the nerve or nucleus is not completely destroyed, are often very greatly benefited by the use of electricity. This is given either by applying one pole to the interior of the larynx, or by applying both poles to the outside of the neck. The intralaryngeal method is exceedingly painful to the patient, can only be borne for a few seconds, and is difficult and tedious of application. Great care must be exercised, especially in abductor paralysis, as the current may produce adductor spasms and occlude the glottis. The application to the neck is much less painful, and can be borne for an indefinite time. The positive pole is usually applied behind the jaw, and the negative pole is passed up and down the larynx and trachea. Either the faradic or the voltaic current may be used. The former is of great value in functional affections of the nerves, but in organic lesions only voltaism is of use. The greater part of the effect is said to be due to the stimulation of the sensory nerves. Massage of the larynx, using the finger and thumb to compress it, is often of very great value, as are also regulated exercises in the use of the voice. If great dyspnœa should occur while the larynx is paralysed, tracheotomy must be immediately performed.

Spasm of the Larynx.

Etiology and pathology.—In this condition there is a sudden contraction of some of the muscles of the larynx, which may last a time, varying from a few seconds to two minutes. The adductors are affected much more frequently than the abductors, since they are not only the more powerful set of muscles, but are also more fully and directly connected with the reflex mechanism of the glottis. In the normal condition, irritation of the glottis, local, reflex, or central, produces closure, by contraction of the adductors.

Children are more liable than adults to spasm of the larynx, because of the greater irritability of their nervous system, and among children rickets is a strongly predisposing condition. In both adults and children, local affections of the larynx and trachea and remote affections in the nervous, digestive, or generative systems may be immediate causes. The attacks come on most frequently during the night, when the reflexes are freed from the control of the cerebral centres, and are more apt to occur in the colder months of the year, from October to May.

Symptoms.—There are several varieties of the spasm, differing in their cause and their symptoms. Laryngismus stridulus is the name given to the simple spasm of the glottis which is common in rickety children. It is doubtful how far the spasm is connected with tetany, the convulsive disease which appears in the same class of cases, but the two conditions are sometimes associated, and are probably due to a common cause, namely, the excessive irritability of the nervous system. There is no doubt that laryngismus differs merely in degree from the general convulsions which occur so readily in rickety children. The spasm seldom appears before the age of 4 months or after 2 years. The immediate cause may be a fright, a painful impression, or some digestive disorder. Sometimes no cause is apparent. The child turns suddenly pale or livid; there are violent inspiratory efforts, involving the extraordinary muscles of inspiration, but no air passes the glottis for a few seconds, or even a minute. Then the spasm relaxes, the air enters with a sudden harsh "crow" or stridor, and the child is apparently well again. If the spasm lasts for more than two minutes the child may die asphyxiated.

The paroxysmal laryngeal spasm of adults is evidently akin to asthma. The attacks come on at night during sleep. The patient wakes suffocating, breathes with the utmost difficulty and with a loud, harsh, laryngeal stridor, which lasts for a few minutes. In severe cases the patient appears to be at the point of death. These attacks may recur at intervals for years. They form the laryngeal crises of locomotor ataxia. Gowers states that they have been known to take the place of attacks of migraine.

In epilepsy the onset of the fit is by a spasm of the larynx producing the well-known "cry"; and in hydrophobia there is well-marked spasm of the glottis. In hysteria the laryngeal spasm may be paroxysmal, or, more rarely, continuous. There is loud inspiratory and expiratory stridor, accompanied by characteristic hysterical rapid breathing. It is sometimes difficult to distinguish the condition from hysterical paralysis of the adductors, but in the latter case inspiration is noiseless, and there is usually paralysis or anæsthesia elsewhere. Spastic aphonia is a functional spasm of the larynx, brought on by the attempt to speak. At first it is impossible for the patient to speak at all, and when he does so the sound of the voice is altered, until by a strong effort he overcomes the difficulty. In other respects the larynx is quite normal; it is only when speech is attempted that the spasm comes on. The condition is thought to be allied to writer's cramp. Gerhardt quotes the case of a man who in earlier days had had writer's cramp, and who at the age of 50 learned to play the flute. The attempt to blow the flute brought on a spasm of the larynx and an involuntary voice sound.

Treatment.—The treatment of laryngismus stridulus is of some importance. During the attack, if asphyxiation threaten, the phrenic nerve may be faradised, or the larynx catheterised, or the chest compressed to produce expiration. If the child is already suffocated, these measures, combined with insufflation of air through the tube in the larynx, may be continued for some time, as in other cases of asphyxia. Between the attacks, sedatives (belladonna, morphine, chloral) may be given, or bromide in small doses. The general health and any remote morbid conditions should be attended to in order to remove the cause of the spasm. The rachitis should be treated by the usual remedies. In adults the treatment does not need to be so active. Stimulation of the skin by cold water or mustard may check the attacks, or the pharynx and larynx may be painted with cocaine. Chloroform may be inhaled in very acute cases. As in children, any exciting cause should be looked for and removed.

The spasm which occurs in epilepsy, hysteria, and tabes must be treated as the disease indicates.

THE ELEVENTH NERVE.

The nerve may be divided into two parts: an external division, or spinal accessory proper, which arises from the spinal cord, from the first to the sixth cervical segment, and which supplies the sterno-mastoid and the greater part of the trapezius muscles; and an internal or vagus accessory, which takes its origin in the lower part of the medulla, joins the vagus nerve, and sends, through it, inhibitory fibres to the heart, motor fibres to the levator palati, azygos uvulæ, some of the constrictors of the pharynx, and the intrinsic laryngeal muscles. Some authors deny that it supplies the larynx.

PARALYSIS.

Etiology and pathology.—The nerve may be affected at its origin from the cord or medulla, within the vertebral canal, at the foramen magnum, or in its extracranial course. The nucleus may be affected by the extension upward of chronic diseases of the cord, such as progressive muscular atrophy, amyotrophic lateral sclerosis, locomotor ataxia, syringomyelia, and by syphilis. Within the vertebral canal the nerve may be injured by caries, tumours, fractures or dislocations of the cervical vertebræ. At the foramen magnum it may be compressed by meningitis or tumours. Extracranially it may be injured by gunshot wounds, stabs, or operations; by pressure of tumours, enlarged or suppurating glands; and it may be affected by rheumatic neuritis from exposure to draught or wet cold.

Symptoms.—When the spinal accessory proper or external branch of the nerve is alone paralysed, only the two muscles which it supplies are affected, namely, the trapezius and sterno-mastoid. The extent of supply to the trapezius appears to vary in different cases. This muscle is usually said to act in three different parts—the upper acting on the clavicle and tip of the acromion process (the *ultimum moriens* of Duchenne), the second on the acromion and outer portion of the tip of the scapula, the third on the inner part and base of the scapula. These parts, which do not act alike, may be paralysed together or separately.

Unilateral paralysis.—In a typical case of unilateral paralysis, the head may be slightly inclined to the paralysed side (though this is not admitted by Gowers), and the chin may be somewhat tilted up. The sterno-mastoid muscle on the affected side is atrophied, and may be reduced to a thin, flabby cord, with corresponding flattening of that side of the neck. Movement of the head towards the sound side is impaired.

When the trapezius is paralysed as a whole, the shoulder on the affected side droops downward and forward, and the tip of the acromion is farther from the middle line than in the normal condition. The curve from the mastoid to the tip of the shoulder is much more concave than on the healthy side, the upper part presenting almost a vertical line from the occiput. The scapula is displaced outwards, and its vertebral border is oblique, the superior extremity being farther out than the inferior. When the arm is raised, the scapula moves outward as a whole; but, in contrast to the position it assumes when the serratus magnus is paralysed, it remains closely applied to the thorax. When the attempt is made to raise the shoulder, the affected side does not rise so high as the other, and the levator anguli scapulæ is seen standing out strongly. The arm can be raised above the horizontal plane, but has not nearly such extensive upward movement as the sound arm, even although the deltoid and serratus magnus are healthy. The upward movement is more extensive when the first or clavicular part of the trapezius is spared.

Bilateral paralysis.—In bilateral paralysis the scapulæ cannot be approximated towards the middle line owing to paralysis of the lowest part of the trapezius, and when this movement is attempted the rhomboids and the levatores anguli scapulæ act and bring the lower part of the vertebral borders of the two scapulæ together, but leave the upper parts widely separated. The forward projection of the clavicles causes the chest to be markedly deepened, and the hollows above the clavicles are accentuated. The spinal column is curved, and the contours of the sterno-mastoid and trapezius are lost, owing to their atrophy. The head is kept in the middle line, tends to fall forward, and can hardly be moved from side to side, especially if the chin be raised. The electrical reactions vary according to the severity of the lesion, from a mere quantitative diminution to the reaction of degeneration.

When the vagus accessory or external branch of the nerve is paralysed, both palate and larynx are affected on the same side, and the pulse rate is accelerated. The palate is broader on the affected side, hangs lower down, and the uvula is drawn to the sound side. The voice is nasal, and, when the larynx is affected, is also hoarse and whispering. There is difficulty in swallowing; many attempts, accompanied by movements of the head, being necessary to convey the bolus to the pharynx. Choking, coughing, and regurgitation of fluids through the nose are common. When the larynx is affected, the crico-arytenoideus posticus is first and most completely paralysed. The vocal cord on one side is stationary in the middle line, and may be thin and atrophied; the sound cord may encroach upon the opposite side during phonation. The voice is low and hoarse. Loud, forcible coughing is impossible. The acceleration of the heart-beats in a case of unilateral paralysis of the cardiac fibres of the accessory nerve is not very marked, the rate usually rising to 80 or 90. In bilateral paralysis it is more marked, rising as high as 100 beats per minute.

Diagnosis.—The diagnosis in the case of paralysis of the accessory nerve is determined by careful examination of the muscles it supplies, the position and power of movement of the head and shoulders, and the condition of the palate and the larynx. It can be distinguished from contracture of the sterno-mastoid by the free passive movement present in the neck, and from contracture of the rhomboids by the free passive movement of the scapula, and the position of the shoulder. In contraction of the rhomboids the outer angle of the scapula is at the normal height, or even raised. In idiopathic muscular atrophy the sterno-mastoid is usually normal, as are also the muscles supplied by the internal branch of the accessory nerve, but other muscles of the shoulder girdle are involved. There are also some cases of partial or complete congenital defect in the trapezius which might mislead, but in such cases the sterno-mastoid is always normal. The electrical reactions would also point to the position and nature of the lesion.

The position of the lesion may be determined by the muscles affected. If the trapezius is paralysed alone, the nerve must be injured after it has passed through the sterno-mastoid. If the sterno-mastoid is also affected, the trunk of the external branch must be injured, and when the larynx and palate are also paralysed the lesion must be intracranial. In some cases the palate and larynx are affected alone and in varying degree, a condition found in diseases of the medulla or meninges affecting the grey matter or the roots at their point of emergence. In such cases some of the other nerves are also usually implicated. When the lesion is supranuclear the sterno-mastoid and the clavicular part of the trapezius are said to escape paralysis.

Prognosis.—When paralysis of the spinal accessory nerve is due to one of the chronic progressive diseases of the cord, the prognosis is very unfavourable. The paralysis is usually progressive and complete, and recovery is almost impossible. In other cases, however, when the paralysis is due to syphilis, compression of the nerve, or neuritis, the prognosis is favourable or unfavourable according to the severity of the lesion and the possibility of its removal.

Treatment.—When the paralysis forms one of the symptoms of a disease, the treatment is that of the disease. When syphilis is present, anti-syphilitic treatment must be employed, and in the case of tumour or caries surgical interference may be necessary. The nutrition of the muscles should be maintained, especially in rheumatic neuritis, by electricity, either the galvanic or faradic current, and their faulty position may be improved by orthopaedic measures.

SPASM OR TORTICOLLIS.

The forms of wry-neck, or torticollis, which result from hysteria or from a lesion in the cervical vertebræ, do not come under consideration here, so that only the true wry-neck will be described. This appears in two forms, as congenital and as spasmodic torticollis.

Congenital wry-neck is thought to be due to traction on the head during delivery, or to oblique position of the head within the uterus. It is not visible at birth, or for the first few years of life, while the neck is short and fat. In such cases the muscle is short and firm, showing a considerable degree of atrophy, which may be confined to the sterno-mastoid, or may involve the uppermost fibres of the trapezius. It almost invariably affects the right side only. The head is kept bent to the affected side, and cannot be turned towards that side. The wry-neck in such cases is easily cured by tenotomy of the sterno-mastoid, but it is frequently associated with facial asymmetry, due to defective development of one-half of the face, which becomes more marked as the child grows older. The same condition may occur in adults, after injury followed by cicatricial contraction of the muscle.

Spasmodic torticollis is a term which includes active spasm of any of the muscles of the neck. It may be tonic or clonic, unilateral or bilateral, and may affect the muscles in the front or the back of the neck, the latter being known as retrocollic spasm.

Etiology and pathology.—In this country it is said to be much more common in women than in men (Gowers). The great majority of cases occur between 30 and 50, but a few occur before and after that age. In the rare cases in which it appears during infancy it does not last more than a few months. Neurotic heredity plays a prominent part in the causation. Epilepsy, insanity, habit-spasm, or torticollis itself may be present in the patient's family, or may have affected himself previously. Debilitated conditions or depressing emotions favour its occurrence. Injury to the neck, such as a blow, or a fall in which the neck is strongly bent, may give rise to spasm, or exposure to cold may induce an attack. Excessive use of the muscles, or frequent movement in one direction, as in some occupations (printing, weaving), may also bring it on. In some cases no cause could be found, and the patient seemed in perfect health when attacked. Paralysis of the opposite muscle has little or no effect in producing torticollis.

No definite morbid condition has as yet been found to account for spasmodic torticollis. The starting-point is not the muscles, since these are quite healthy, and the question is whether it is the spinal centres or the cerebral centres. It is possible that the lesion may be in either. When a group of muscles which act together physiologically, but are anatomically separate, are affected, the lesion would probably be found in the cerebrum, whereas when several muscles are affected which have no physiological connection, but are supplied by nerves from the same level in the cord, the chances are that the lesion is spinal. It must be borne in mind, however, that nerve cells supplying muscles that are functionally associated may have connections within the cord, and thus a lesion there might affect a group of muscles that habitually act together. As to the changes in the nerve cells nothing is known, except that there is some form of spontaneous discharge from them, causing the spasm.

Symptoms.—The symptoms are pain, and tonic or clonic spasm usually confined to one side of the neck. The pain may precede or accompany the attack, and may be dull and gnawing, or acute and neuralgic in character. It may be in the neck, behind the ear, or in the arm. The onset of the spasm is slow. It ceases during sleep and rest, but is increased by exertion.

In the tonic spasm the head is held with the chin raised, the occiput drawn to the affected side, and the face rotated to the sound side. The sterno-mastoid is the muscle most commonly affected, but the upper division of the trapezius may also be involved, in which case the head is much more depressed towards the affected side.

Clonic spasm is much more distressing in its nature. It may affect only one muscle, which is almost invariably the sterno-mastoid on one, or, rarely, on both sides, or it may affect a group of muscles. The upper fibres of the trapezius on the same side, or the splenius on the opposite side, are very often involved, and in rarer cases the platysma myoides, or the recti and obliqui. In a typical case in which the sterno-mastoid alone is affected, the spasm of the muscles draws the mastoid process towards the sterno-clavicular articulation on the affected side, so that the chin is tilted outwards and upwards, and the whole face turned to the sound side. When the upper fibres of the trapezius are also included in the spasm, the occiput is drawn more to the affected side, downwards and backwards, and the shoulder is raised, the scapula being rotated. The middle or lower parts of the trapezius are rarely involved. When the trapezii and splenii on both sides are contracted, the head is drawn backwards, producing retrocollic spasm. The clonic spasm usually recurs every few moments, or may be practically continuous. The muscles become hypertrophied, and pain or fatigue may be felt in them. The electrical reaction may be normal, or the excitability may be excessive. There is a tendency for the spasm to extend to the arm, the face, or even the muscles of mastication. The arm may be affected on the same side or on the opposite side, the face on the same side.

The course of the disease varies. In a very few slight cases the spasm may cease spontaneously in a few months or years, but in the majority of cases it begins in one or two muscles, spreads to others, and increases in intensity for a few years. It then becomes stationary, persisting for the rest of life, or it may cease for a time, and then return. It does not shorten life, but is very annoying and burdensome to the patient.

Diagnosis and prognosis.—There is seldom any difficulty in diagnosing torticollis, nor in distinguishing between the true and false form. In true torticollis the face is turned away from the affected side, whereas in the spurious forms it is turned towards it. Slight limited retrocollic spasm may be mistaken for simple tremor, but the movements in tremor are seldom so violent, and contractions of the frontal fibres of the occipito-frontalis muscle are not so marked as they are in retrocollic spasm.

Hysterical spasm in the neck may simulate torticollis. The hysterical nature of the affection may be suspected in women under 30, or when the movements spread from the neck to the trunk. It may, however, be impossible to determine the nature of the spasm.

In all severe and extensive cases the prognosis as to recovery is very grave. It is more favourable when the spasm is limited or shifting, and when it occurs in the first half of adult life.

Treatment.—Torticollis may be treated by drugs, by mechanical supports, or by surgical interference. The drugs used may be nervine tonics, such as valerianate of zinc and asafœtida, or sedatives, especially bromide of potassium, conium, Indian hemp, chloral, and morphine or opium. Unfortunately, the spasm usually returns when the drug is discontinued. Morphine injected hypodermically sometimes has a permanent influence, but should be used as a last resource, so great is the tendency to the formation of a habit. It should be used daily for several months, the dose being gradually increased to one-half or one grain a day, when in many cases it has completely stopped the spasm. The difficulty is the discontinuance of the drug, as the patients become so habituated to it that they cannot do without it, and find it difficult or impossible to break off the habit. It should therefore be employed only when all other efforts to stop the spasm have failed.

Electricity is of very little use. A weak voltaic current has been employed for its sedative action, the positive pole being placed just below the occiput, and the negative on each affected muscle in succession for five or ten minutes. Counter-irritation over the affected muscles may do good, a blister being the most effectual.

The range of movement during spasms may be lessened by the use of mechanical supports. The best form is a rigid rod up the back of the neck and head, with a small pad projecting on the affected side, which checks the downward movement of the occiput.

The only form of surgical interference that is of permanent use is the division of the spinal accessory nerve before it enters the sterno-mastoid. The nerve is cut and half an inch removed to prevent reunion. The muscles it supplies become paralysed and atrophy, and there is slight loss of power of movement of the head and shoulder, but the relief gained by the freedom from spasm more than compensates for this. Division of the tendon of the sterno-mastoid in the spasmodic affection is worse than useless, and nerve-stretching has only a temporary effect.

THE TWELFTH NERVE.

PARALYSIS.

Etiology.—The lesions which produce paralysis of the tongue may be classified according to their position. Those which occur above the nucleus, *supranuclear* lesions, may be situated on any part of the motor tract between the nucleus and the cortex of the lowest part of the ascending frontal and hindmost part of the third frontal convolutions. The most frequent lesions here are hæmorrhage, softening, tumour, and traumatism. Those which occur at the nucleus, *nuclear* lesions, may be hæmorrhage, degeneration, softening due to vascular occlusion or inflammation, bulbar paralysis, locomotor ataxia, syringo-myelia, general paralysis, or tumour. In the third class of cases, the *infranuclear*, the lesions may be either within the medulla or outside it. Within the medulla, tumour and softening are the most common lesions. Of the extra-medullary lesions, the most important are meningitis (simple or syphilitic), tumours at the base of the brain, tuberculous, syphilitic, or carcinomatous disease of the bones at the base of the skull, or upper cervical vertebræ, or dislocation at this point. In one case a hydatid cyst in the anterior condyloid foramen produced paralysis of the hypoglossal nerve. Penetrating and gunshot wounds and local cellulitis may affect the nerve. Some cases of rheumatic neuritis and congenital atrophy of the nerve have been noted. Gowers also refers to the fact that hemiatrophy of the tongue is sometimes associated with hemiatrophy of the face.

Almost any of the causes which produce unilateral paralysis of the tongue may give rise to the *bilateral* forms, the exception being the peripheral lesions. No purely peripheral lesion has been known to result in paralysis of both hypoglossal nerves. A bilateral paralysis of the tongue constitutes an important feature in bulbar and pseudo-bulbar paralysis, and also appears in amyotrophic lateral sclerosis and chronic anterior polio-myelitis when these diseases extend to the medulla.

Symptoms.—**Unilateral.**—When the paralysis is unilateral and the lesion nuclear or infranuclear, there are, as a rule, no subjective symptoms. There is no loss of taste, no alteration of common sensibility, and little, if any, impairment of speech, chewing, or swallowing. When the tongue lies in the mouth, its sound side may sometimes appear shorter than the other, owing to the "tone" of the healthy muscle, whereas the base of the affected side may be higher, from lack of tone in the genio-hyoglossus and hyoglossus muscles. When protruded, the tongue tends towards the paralysed side, the raphe being concave to that side. This half of the organ is atrophied, being thin, furrowed, and wrinkled, and sometimes covered with a thicker fur than is the sound side. Fibrillary tremors are constantly present in it. If the lesion is above the nucleus, the atrophy and fibrillary tremors do not usually occur.

If the nerve is injured between its junction with the loop from the cervical plexus and the point at which the descendens noni is given off, the depressors of the hyoid bone and thyroid cartilage are atrophied, so that the latter structure becomes more prominent than usual, and is drawn to the sound side during deglutition. The muscles supplied by the hypoglossal nerve respond abnormally to electric stimuli, whether applied directly to them or indirectly through the nerve. The alteration is usually quantitative, but may be qualitative, so that, according to the severity of the lesion, the reaction of degeneration is partial or complete.

Bilateral.—In bilateral paralysis the subjective symptoms are much more marked. Speech is very imperfect, while chewing and swallowing can only be performed with difficulty, owing to the inability to move the food about within the mouth, or to use the tongue in forcing the bolus back into the grasp of the palatal muscles. Taste is impaired by the inability to move the food within the mouth, but sensibility is, of course, unaffected. Saliva tends to accumulate in the mouth. The tongue lies almost motionless in the middle line, and cannot be protruded at all, unless the paralysis is incomplete, in which case it may be projected as far as the lips, or may be merely tremulous on protrusion. It is much reduced in size, being thin, flabby, furrowed on its upper surface, and notched at its edges. It shows well-marked fibrillary tremors over its entire surface.

Diagnosis.—Interference with the hypoglossal nerve may be assumed when there is a history of injury to the neck followed by atrophy and paralysis of the half of the tongue on that side. In other cases, when the lesion is due to some disease of the cord and medulla, such as tabes, syringo-myelia, bulbar paralysis, etc., there will be other symptoms of the disease present. In cases of syphilitic affections of the bones or membranes there will be evidence of syphilis, and, as a rule, other nerves will be affected at the same time.

The situation of the lesion must be determined by the presence or absence of paralysis elsewhere, and by the trophic condition of the tongue. If the lesion be supranuclear, there will probably be some other paralysis on the same side of the body, the tongue will not be atrophied, and its muscles will not show the reaction of degeneration. If the nucleus be affected, the paralysis is usually double and affects the lips very noticeably; while, if the lesion be infranuclear, but still within the medulla, there is usually paralysis in some part of the opposite side of the body. When the nerve is affected after leaving the medulla, other nerves, usually the vagus and spinal accessory, are, as a rule, also affected. In all nuclear and infranuclear lesions the tongue is atrophied and the reaction of degeneration is present. The palate may be paralysed on the same side as the tongue when the nerve is injured at the point of emergence from the medulla, from implication of the spinal accessory nerve.

The position of a lesion in the peripheral course of the nerve is determined by the presence or absence of paralysis of the palate or the depressors of the hyoid bone, or by the concomitant paralysis of the sternomastoid and trapezius. The following table may express the various conditions in a concise manner:—

- Unilateral
  - Without atrophy
    - Supranuclear—is generally associated with hemiplegia or hemiparesis of the same side.
  - With atrophy
    - Nuclear—rare: lips generally also involved.
    - Infranuclear
      - Medullary
        - Associated with paralysis of opposite arm and leg. Tongue protruded to side opposite that of paralysed limbs.
      - Extramedullary
        - Associated with paralysis of corresponding half of palate and larynx.
      - Extracranial
        - Sometimes associated with paralysis of palate and larynx. Occasionally with paralysis of depressors of hyoid and thyroid, or with paralysis of trapezius and sterno-mastoid.
- Bilateral
  - Without atrophy—pseudo-bulbar paralysis.
  - With atrophy
    - Nuclear—Also associated with paralysis of lips and throat.
    - Medullary
      - Associated unilateral or bilateral paralysis of upper and lower limbs.

Prognosis.—The prognosis in cases of paralysis of the hypoglossal nerve is very unfavourable when the lesion is in the brain, the medulla, or the cord, more especially if it forms part of one of the chronic progressive diseases of these regions. In such cases complete recovery is almost impossible. Where the nerve is injured on one side only, the prognosis is more favourable if the nerve be only indirectly crushed and not directly injured. When the paralysis is due to syphilis, the prognosis may be fairly good, unless the nerve itself or its centre has been destroyed.

Treatment.—The treatment of the paralysis of the hypoglossal nerve must be directed to the diseases which caused it. Most benefit may be expected from antisyphilitic remedies when these are specially indicated. If it is thought desirable to use electricity, the faradic and galvanic currents may be employed. Gowers recommends that the pole applied to the tongue should be connected with a tongue depressor with a wooden handle, and insulated by sealing-wax to prevent contact with the lips. Erb applies the anode to the neck, and the cathode to the motor point at the angle of the jaw. The currents, whether galvanic or faradic, should be strong enough to induce movements of deglutition.

SPASM.

Etiology and pathology.—Spasm of the tongue hardly ever occurs alone; it generally forms a symptom of some other general nervous disease, such as hysteria, chorea, epilepsy, insanity (hypochondriasis, melancholia), or is caused by reflex irritation. In epilepsy the protrusion of the tongue coincidently with spasm of the masseters during the clonic stage of a fit may lead to its being bitten. In chorea it may be jerked out and retracted spasmodically and irregularly. In hysteria and in conditions of mental impairment it may be protruded in a rigid manner, or alternately protruded and retracted. A peculiar condition, termed aphthongia by Fleury, induced by emotion, and rendering articulation impossible, is due to spasm of the tongue. So are also some forms of stuttering. When spasm of the tongue is associated with spasm of the lower part of the face, it may be either functional in origin or due to irritation of the special centres in the motor cortex. It may be produced reflexly by irritation of one of the branches of the fifth nerve (supplying the teeth, gums, tongue, nose), the glosso-pharyngeal (tonsils, pharynx), or even by the occipital nerve. It may also be caused by basal meningitis. The disease occurs most commonly between 16 and 40. It is rare in old age and not common in childhood, except as a symptom of an organic lesion at the base of the brain or in the cerebrum.

Symptoms.—The spasms may be tonic or clonic in character. In tonic spasm the tongue may be rigidly fixed against the floor of the mouth, the upper teeth, or the hard palate. When the spasms are clonic, the paroxysms may occur daily or several times a week. They seldom occur during sleep, but when they do so may waken the patient. The spasms may recur thirty to fifty times a minute, and may forcibly protrude the tongue from the mouth, or may project it against the hard palate or against the closed teeth.

During the attacks, speaking and swallowing are almost impossible, and the tongue may be so strongly retracted that the epiglottis is pressed down on the larynx and suffocation appear imminent. The spasm may be without apparent cause, or may be induced by over-exertion or emotion. It may be preceded by an aura, either a feeling of numbness, or tingling in the tongue, or of tension in the larynx. Yawning and excessive lachrymation or salivation may accompany the spasm. Consciousness may be lost if the spasm is epileptic in origin.

Diagnosis and prognosis.—The diagnosis seldom presents any difficulty. The affection of the tongue is very marked, and the associated conditions are generally very evident, whether these are due to functional or to organic disturbances. The disease may run a course of several months, and greatly weaken the patient by interference with nutrition and sleep; but except when the cause is an organic one, it generally yields to appropriate treatment. When the tongue is drawn back so as to depress the epiglottis, suffocation may be threatened.

Treatment.—The treatment must be directed to the causal disease, and will generally require the use of tonics, such as iron, arsenic, and antispasmodics. Sources of reflex irritation should be sought for and removed. The galvanic current sometimes renders signal service, especially when the anode is applied below the angle of the jaw.

THE PHRENIC NERVE.

The phrenic nerve is formed by the junction of a branch from the fourth cervical nerve and a smaller branch from the fifth. It passes down the neck in front of the scalenus anticus, enters the thorax between the subclavian artery and vein, passes across the root of the lung, and pierces the diaphragm, to be distributed to its under surface.

PARALYSIS.

Etiology and pathology.—In its course it may be affected by disease of the cord or the roots by which it is formed, by disease of the membranes, such as tumour, pachymeningitis, meningitis (syphilitic or tuberculous), or by hæmorrhage. Caries, tumour, fracture, or dislocation of the cervical vertebræ may affect the nerve at its origin, or it may be injured by a wound in the neck, or by the growth of a tumour in this situation. Within the thorax it may suffer compression from enlarged glands, tumour, or aneurysm, and it has been known to suffer in acute or chronic purulent inflammation extending from the pleura or peritoneum to the diaphragm, and injuring the nerve endings within the muscle. It may be affected by neuritis, rheumatic, toxic, or infective (diphtheria, influenza), or may be involved in multiple neuritis. Alcoholic polyneuritis sometimes ends fatally by extension of the disease to the phrenic. The chronic progressive diseases of the cord and medulla, such as progressive muscular atrophy, may also affect the phrenic nerve.

Symptoms.—When both phrenic nerves are paralysed, there is complete immobility of the diaphragm. While the patient is at rest the respiration may be normal or slightly accelerated. During inspiration the epigastric and hypochondriac regions sink inwards from absence of the contraction of the diaphragm, so that the normal expansion of the abdominal wall is lost. And during expiration the opposite movements occur. On exertion there is dyspnœa, the breathing is rapid and the voice faint. Deep inspiration is impossible, and the reflex movements involving the contraction of the diaphragm, such as coughing, vomiting, defæcation, etc., are considerably impaired. If the hand be placed on the abdominal wall and pressed upwards, it will be found that the abdominal viscera can be displaced upwards into the thorax, and the lower limit of the lungs raised a little. Unilateral paralysis of the diaphragm may produce no symptoms, and can only be ascertained by careful inspection. The immobility of the paralysed side is masked by the movement on the other side. No sensory disturbances, beyond the presence of pain behind the sternum and costal cartilages, have been described.

In neuritis of the nerve there is a tender spot on the scalenus anticus, between the two heads of attachment of the sterno-mastoid. The electrical reaction of the nerve may be greatly altered; in some cases the nerve only responds to the galvanic current, and in other cases neither the faradic nor the galvanic current have any effect. The point at which the nerve may be stimulated is a small area at the outer margin of the sterno-mastoid, where the nerve lies in front of the scalenus anticus.

Diagnosis.—The diagnosis of paralysis of the diaphragm is not difficult when both sides are affected and there are signs of a lesion in the cervical region of the cord or spine. The abnormal respiratory movements in the abdominal wall, the dyspnœa, etc., on exertion, clearly indicate the condition. Some difficulty may be found in the case of hysterical patients, especially women, who normally use the diaphragm less in respiration than do men. Careful examination, the attention of the patient being meanwhile distracted, is required. One single diaphragmatic inspiration will decide the question. There may be great difficulty in determining a unilateral paralysis, so slight is the loss of movement. The history is of great importance in all cases, and careful examination for injury or tumour in the neck should be made. The presence of a tender point on the scalenus anticus, just outside the sterno-mastoid, would indicate neuritis of the phrenic. The inaction of the diaphragm in pleurisy or peritonitis may be distinguished from true paralysis by the accompanying pain in neighbouring parts.

The site of the lesion is easily determined as a rule by the presence of a tumour or injury. When the lesion is in the cord, other muscles supplied by the third and fourth cervical nerves are paralysed. When the nerve alone is affected, the paralysis is limited to the diaphragm, unless the condition is part of a multiple neuritis.

Prognosis.—Speaking generally, the prognosis in cases of paralysis of the diaphragm is unfavourable as regards recovery. Although in itself not dangerous to life, so long as the patient remains quiet, it may be so if a morbid process in the lung (bronchitis, pneumonia) should supervene or be previously present. In cases of chronic alcoholism, or chronic disease of the medulla or cord, the prognosis is bad, and also in injury or tumour of the cervical cord or membranes. Paralysis due to hysteria, syphilis, or lead-poisoning usually does well under treatment, and when the condition follows an acute infective disease (diphtheria or influenza), recovery may also take place, though less frequently.

Treatment.—In all cases the cause of the lesion should be treated; wounds should be attended to, and where possible any tumour should be removed. If the condition be due to hysteria, syphilis, or lead-poisoning, the appropriate treatment must be employed. The patient should be kept quiet in bed, no exertion or excitement allowed, and the general nutrition should be maintained by good feeding. Counter-irritation may be applied, in the shape of blisters along the course of the phrenic nerve; and strychnine may be administered subcutaneously.

Electricity has proved of great service. The faradic current should be used, one electrode being placed at the outer border of the sterno-mastoid muscle above the clavicle, and the other on the epigastrium, or over the corresponding half of the diaphragm.

THE POSTERIOR THORACIC NERVE.

The posterior thoracic nerve, or long respiratory nerve of Bell, is formed in the substance of the scalenus medius by branches from the roots of the fifth and sixth cervical nerves. It passes downwards behind the brachial plexus to the side of the thorax as far as the lower border of the serratus magnus, which it supplies by piercing its outer surface.

PARALYSIS.

Etiology and pathology.—The long and exposed course of the nerve renders it liable to injury from external pressure, as by carrying a heavy weight on the shoulder; from punctured or gunshot wounds in the neck or axilla; or from severe falls or blows on the shoulder. It may also be injured by severe muscular exertion, such as carrying a great weight, or wielding a heavy hammer. In these cases the lesion is usually a traumatic neuritis set up by the violent contraction of the muscles through which the nerve passes. Neuritis due to cold (rheumatic) or forming a sequel to acute infective disease, has been observed. Probably the paralysis which occurs after parturition is due either to violent muscular exertion or to exposure of the neck to cold during labour.

In the progressive bulbar and spinal diseases, such as infantile paralysis or progressive muscular atrophy, the posterior thoracic nerve may be affected in common with other nerves. From what has been said, it follows that the lesion is most common in muscular workers, and therefore occurs very much more frequently in men than in women. For the same reason it is usually found on the right side, in adults between 25 and 40. It is rarely bilateral, except when due to cerebral disease, or to carrying a heavy weight first on one shoulder and then on the other.

Symptoms.—The symptoms are due to the loss of power of rotation and fixation of the scapula caused by paralysis of the serratus magnus. While at rest there may be very little displacement. In most cases the lower angle of the scapula while at rest is rotated inwards and upwards, and is more prominent than usual, owing to the absence of fixation by the serratus magnus, and the upward and forward pull on the bone exerted by the combined action of the levator anguli scapulæ and the muscles attached to the coracoid process. The latter muscles, and the weight of the arm, tend to pull the shoulder down, and thus aid the inward and backward displacement of the inferior angle. The vertebral border is nearer the middle line than usual. During forward and upward movement of the shoulder the effect of the paralysis is very marked. The patient, as a rule, cannot raise the arm higher than the horizontal position. When thus raised, the vertebral border of the scapula is brought abnormally near the middle line, and projects backward from the thorax, especially at its inferior angle, so that a deep furrow is produced, into which the hand can be placed, and thus the ventral aspect of the subscapular muscle partly palpated. The middle fibres of the trapezius, the posterior fibres of the deltoid, the infraspinatus, and the teres major and minor, take on the function of the serratus magnus, so that a certain amount of elevation above the horizontal is accomplished; but this and other movements are much weakened.

In bilateral paralysis the appearances just described are present on both sides. When both arms are raised to the horizontal, the two scapulæ may touch each other in the middle line, forming a complete arch. The respiratory movements are not noticeably affected. Sensory disturbances are limited to the acute pain which precedes and accompanies the neuritis. Atrophy of the muscle takes place in long-standing cases, and is most noticeable at the insertion on the ribs, where the digitations are seen to be small and thin.

Prognosis.—The prognosis is favourable as regards ultimate recovery in cases where the neuritis is due to injury, or to rheumatic or infective disease, but the duration of the paralysis varies greatly. It may recover within six weeks, but may last for months or a year, or may be permanent. When marked reaction of degeneration is present, or when electrical excitability is quite lost, the prognosis is grave. In the chronic diseases of the cord and medulla the prognosis is absolutely unfavourable.

Treatment.—In the early stages, diaphoretic treatment may be tried and anti-neuralgic drugs given for the pain. Little benefit is derived from either. The arm should be supported so as to raise the shoulder and put the muscle at rest, and no movement involving the serratus should be allowed. In later stages counter-irritation may be applied over the scalenus, and electricity employed. When the neuritis is slight the faradic current may be sufficient; but in graver cases galvanism must be used. The cathode should be placed in the supraclavicular fossa, the anode on the side of the neck, and a constant current passed for three to five minutes; the anode should then be moved up and down over the serratus between the latissimus dorsi and the pectoralis major. Much benefit has been derived from persistent massage. In incurable cases a broad firm bandage round the chest fixing the scapula to the thorax has been said to answer well.

THE SUPRASCAPULAR NERVE.

The suprascapular nerve rises from the trunk formed by the fourth, fifth, and sixth cervical nerves, but its fibres are derived from the fourth and fifth only. It supplies the supraspinatus and infraspinatus muscles.

Isolated paralysis of the nerve is rare, but it may be injured, either alone or along with the circumflex nerve, by dislocation of the humerus, by falls on the shoulder, or by the pressure of heavy weights carried on the shoulder. In some cases it is involved in injury of the upper part of the brachial plexus, when it is usually the last to recover.

The symptoms produced by injury to the nerve are limited to paralysis of the two muscles it supplies. The loss of the infraspinatus is most important, since the power of external rotation of the arm is interfered with and many movements thus impaired. Writing becomes difficult from loss of power of outward rotation of the arm as the pen is moved along the line. The posterior fibres of the deltoid take on the function of the paralysed infraspinatus, and may become greatly hypertrophied. It will be noted also that the inferior angle of the scapula rotates upwards and inwards from the unopposed action of the rhomboids, and that the supra- and infraspinous fossæ show depressions from atrophy of the muscles. Paralysis of the supraspinatus is unimportant. It merely supplements the deltoid, so that when both are paralysed the head of the humerus falls from the shoulder more than if the deltoid were paralysed alone. When the supraspinatus is paralysed alone, the humerus is less firmly fixed to the shoulder than normally, and the deltoid is more readily exhausted. Sensation may be unaffected, but frequently there is some anæsthesia over the scapula.

THE CIRCUMFLEX NERVE.

The circumflex nerve arises from the posterior cord of the brachial plexus, its fibres being derived from the fourth and fifth cervical roots. It supplies motor fibres to the deltoid, the teres minor, and sensory fibres to the skin over the lower part of the deltoid.

The nerve may be affected by dislocation, or by fracture of the upper part of the humerus, or by severe falls or blows on the shoulder. The pressure of tumours of the clavicle or in the axilla may paralyse it, or the pressure of a crutch used constantly. It is also frequently paralysed in miners, by the constant pressure on the deltoid from lying upon the left side when using the pick while at work. Simple neuritis (rheumatic paralysis) is rare; but the nerve may be affected in multiple neuritis, acute infective diseases, and toxic conditions, such as diabetes and lead-poisoning. In acute rheumatism, inflammation may spread directly from the joint to the nerve. The fibres of the nerve may be included in disease of the upper part of the plexus, and both nerves and muscles are very often affected in disease of the central and peripheral nervous system, in hemiplegia, progressive muscular atrophy of the scapulo-humeral type, general lead-poisoning, and hysterical paralysis.

When the deltoid is paralysed, the arm cannot be raised from the side. A few of its anterior fibes, which are supplied by the anterior thoracic nerve, may contract; but even with the aid of the coraco-brachialis the movement they produce is very slight. When the condition has persisted some time, the muscle atrophies, and the outline of the shoulder is greatly altered in consequence, its rounded appearance being lost and the sharp angle of the acromion process made visible. The paralysis of the teres minor has little effect. Sensory disturbance is present as anæsthesia over the lower part of the deltoid in the area supplied by the cutaneous branches of the circumflex, and in the early stages there may be acute pain in the shoulder.

The electrical reactions vary with the severity of the lesion. The diagnosis can be easily made from the immobility of the arm and the atrophy of the muscle. It may be difficult sometimes to distinguish paralysis of the muscle from ankylosis of the shoulder in stout subjects where the atrophy is marked; but the fact that in ankylosis the scapula moves with the arm in passive movement, serves to distinguish the two conditions.

THE MUSCULO-CUTANEOUS NERVE.

The musculo-cutaneous nerve arises from the outer trunk of the brachial plexus, its fibres being derived from the fourth and fifth cervical roots. It supplies motor fibres to the biceps, coraco-brachialis, and part of the brachialis anticus, and sensory fibres to the skin on the radial and part of the inner and outer surfaces of the forearm, as far as the ball of the thumb and the palm.

Isolated paralysis of the nerve is rare, but it is very commonly paralysed with other nerves in disease of the brachial plexus, dislocations of the shoulder, extirpation of a tumour in the supraclavicular fossa, or in incised or gunshot wound of the upper arm. It is also affected in Erb's paralysis.

When the muscles supplied by the musculo-cutaneous nerve are paralysed, the forearm hangs down in extension, and cannot be flexed upon the arm. If the forearm is supinated, there is absolutely no power of flexion at the elbow, but when it is in pronation a slight amount of flexion is obtained by the action of the supinator longus. There is anæsthesia on the radial side of the forearm in the region supplied by the nerve.

THE MUSCULO-SPIRAL NERVE.

The musculo-spiral nerve rises from the posterior cord of the brachial plexus, deriving its motor roots from the lower four cervical nerves. It supplies the triceps, the two supinators, and all other muscles on the back of the forearm. It gives sensory branches to the skin on the radial side of the back of the hand, the back of the thumb, the index finger, and one-half of the middle finger. The dorsal aspect of the terminal phalanges of the thumb, and the first, second, and third fingers are variably supplied—sometimes by the radial and sometimes by the median nerves.

PARALYSIS.

Etiology and pathology.—The most common cause of paralysis of the musculo-spiral nerve is pressure, to which it is greatly liable in its long, exposed course. The pressure usually occurs during heavy sleep, especially when the sleep is due to alcoholic excess. Sleeping on a hard bench or on the ground with the head resting on the arm, or sleeping with the arm hanging over a chair or couch, is specially apt to produce the condition. One characteristic of the paralysis due to pressure during sleep is the absence of paralysis of the triceps, since the nerve is compressed below the point at which the branch to the triceps is given off. The nerve is often injured higher up by the pressure of a badly-padded crutch, or by dislocation of the head of the humerus. In fractures of the humerus the ends of the bones may grasp the nerve, or it may be included in the growth of a callus, or may be affected by tumour or necrosis of the humerus, or injury to the arm by gunshot or incised wounds. Injuries to the elbow joint sometimes involve the nerve, and its branches may be implicated in injuries to the forearm. Ligatures compressing the arm, or tying the arms behind the back, may cause paralysis, and in Russia it is often caused in infants by the habit of binding their arms to their sides before laying them down to sleep. Violent contraction of the triceps, such as occurs in throwing a stone or pulling on tight boots, may also injure the nerve. Cold and exposure to wet have been said to produce a rheumatic neuritis, and after acute infective disease or toxic conditions paralysis may occur. Hypodermic injection of ether into the forearm sometimes gives rise to a neuritis which may extend upwards along the nerve.

Symptoms.—The symptoms of paralysis of the musculo-spiral nerve depend upon the site of the lesion. When this is in the brachial plexus, all the muscles supplied by the nerve are paralysed, and there is loss of power of extension at the elbow, the wrist, and the fingers, combined with loss of power of supination. Complete "wrist-drop" is present, and the fingers cannot be extended at the metacarpo-phalangeal joint. The loss of supination is very serious, from the great inconvenience it causes. Any attempt to grasp an object firmly produces pronation in the forearm, and to counteract this the elbow is pressed against the side and the humerus rotated outwards. In the course of time the pronators may become permanently contracted, and a prominence forms on the back of the carpus due to projection of the synovial sacs from overflexion of the wrist and loss of the support of the extensor tendons. The flexors are much weaker than on the sound side, from loss of the antergic support of the opposing tendons.

When the nerve is injured in the middle of the arm, the triceps escapes, as a rule; and when the lesion is below the nerve to the supinator longus, or if the paralysis is incomplete, the latter muscle escapes also. In sleep paralysis the supinator longus is usually involved, but may escape. The fingers are, as a rule, unequally affected—the first finger being least paralysed, the fourth most so. The loss of sensibility is very variable. The skin of the upper arm is only anæsthetic when the nerve is injured very high in its course, usually by the pressure of a crutch in the axilla. When the injury is lower, there may be anæsthesia of the skin over the thenar eminence, the radial side and the back of the hand as far as the third metacarpal bone, and one or both phalanges of the thumb. Subjective sensations of cold, numbness, and tingling may be felt in the parts. There is usually marked atrophy of the muscles, the paralysed limb being $\frac{1}{3}$ to $\frac{1}{2}$ in. less in circumference than the sound one.

Diagnosis and prognosis.—The diagnosis of paralysis of the musculo-spiral is easy, as a rule. Lead palsy is the only condition for which it is likely to be mistaken, and from this it can be distinguished by the history of a distinct cause, the sudden onset, the limitation to one arm, and the affection of the supinators. The nerve is often affected in the various forms of polyneuritis. The condition of the electrical excitability of the muscles offers the surest guide to the prognosis of the course and duration of the paralysis. In slight cases, where the excitability of the nerve is only slightly altered, the prognosis is good, recovery taking place in a few weeks. Where the middle form of reaction of degeneration is present, the condition may persist for two or three months, severe cases taking six months to recover. The condition rarely becomes permanent. The prognosis in paralysis due to pressure during sleep is more favourable than in that due to severe injury.

Treatment.—The paralysis due to slight compression usually recovers spontaneously, but recovery may be accelerated by the use of the galvanic current for five or ten minutes daily. In the acute stage rest and sedative treatment should be employed. In the more severe cases the galvanic current should also be employed, and in all cases attention should be paid to the posture of the limb, any tension on the nerve being avoided. When the portion of the nerve that winds round the humerus is injured, extension at the elbow should not be permitted, as the contraction of the triceps may injure the nerve further. Counter-irritation and massage are of benefit in the later stages of the condition. Surgical interference is required if the nerve has been divided by a wound, or is compressed by fracture, callus, dislocation, or tumour.

THE MEDIAN NERVE.

The median nerve rises partly from the outer and partly from the inner head of the brachial plexus, and derives its fibres from the four lowest cervical roots. It supplies motor fibres to the pronators, the flexor carpi radialis, the superficial flexor and radial half of the deep flexor of the fingers, the long flexor of the thumb, the intrinsic muscles of the thumb with the exception of the adductor, and the inner head of the flexor brevis, and, lastly, the two outer lumbricales. Its sensory distribution is to the radial surface of the palm (excepting the most external portion of the ball of the thumb), the anterior aspect of the thumb, the first two fingers and the radial half of the third finger. The posterior aspect of the second and third phalanges of the first and second fingers, and the terminal phalanx of the thumb and ring finger have their sensory supply, as already said, usually from the median, but sometimes from the radial branch of the musculo-spiral.

PARALYSIS.

Etiology and pathology.—Isolated paralysis of the median nerve is rare, and, when it occurs, is due to injury to the forearm. The most common cause is a cut in the anterior surface of the forearm, from a fall on or through glass, or a wound from a knife. A gunshot wound in the forearm, or fracture of the bones, or a tumour in the course of the nerve, may also cause paralysis. Pressure from an Esmarch's bandage, or from tying the hands behind the back, may paralyse the median nerve, as also may the pressure of some implement on the palm of the hand, as is sometimes shown in laundrymaids, whose work entails the frequent use of a flat iron, and in joiners or smiths. Higher up the arm injury may be caused by the pressure of a crutch, or by hyper-extension during long-continued narcosis, or by surgical interference in cases of tumour. Over-exertion of the muscles of the forearm has been said to set up neuritis, and the same condition may be found after acute infective diseases, in the puerperal state, or in chronic poisoning by lead or arsenic. It is doubtful whether a rheumatic neuritis ever affects the median nerve. It may, however, be involved in central diseases of the brain and cord, the most common being progressive muscular atrophy, which usually appears first in the small muscles of the hand.

Symptoms.—The symptoms of paralysis of the median nerve vary with the situation of the lesion. When this is in the brachial plexus or upper arm, the paralysis of the nerve is complete. Pronation of the forearm is entirely lost in such cases, or can only be imperfectly performed by the supinator longus, which brings the hand to mid-position, the elbow being flexed and the humerus rotated inwards and abducted. Flexion of the wrist is greatly impaired by the loss of the radial flexor, and when performed in part by the ulnar flexor is accompanied by marked inclination of the hand to the ulnar side. Flexion of the fingers is much weakened, since only their proximal phalanges (which are flexed by the interossei) and the terminal phalanges of the fourth and fifth finger are exempt from paralysis. The action of the interossei tends to over-extend the second and third phalanges as well as to flex the first, so that the fingers, especially the first, remain in a position of over-extension at the last joints. The paralysis of the short muscles of the thumb causes great inconvenience, as it cannot be opposed to the other fingers, but rests in persistent adduction and extension, like the thumb of the monkey.

When the nerve is injured in the lower forearm, the power of pronation and of flexion of the wrist and fingers remains, but the small muscles of the hand which are supplied by the median are paralysed. The condition of sensation varies from complete loss to almost perfect preservation. When impairment is present it is usually most distinct on the palm, but may also extend to the dorsal aspect of the terminal phalanges of the thumb and first three fingers. Atrophy of the muscles, especially in those that form the thenar eminence, is usually marked in old cases. There may be some trophic changes in the areas supplied by the nerve, such as coldness or lividity. Sweating is diminished, and the growth of hair increased. The skin becomes glossy, and is easily destroyed or injured by slight causes, such as bathing with hot water, which may cause a pemphigus-like eruption, or slight contusions which would not injure the normal skin. The nails may grow irregularly or show marked longitudinal grooving.

The electrical reactions are the same as in other paralysis in the arm, and the prognosis also has been already given in dealing with the other nerves. In cases of division of the nerve, recovery is usually rapid if the ends are carefully joined. The diagnosis is easy, the paralysis and atrophy of the muscles of the thumb, and the distribution of the anaesthesia are so distinctive. The history of the cause will usually show whether the lesion is central or peripheral.

Prognosis and treatment.—These are essentially the same as already described in dealing with the other nerves of the arm.

THE ULNAR NERVE.

The ulnar nerve rises from the inner cord of the brachial plexus, and derives its fibres from the eighth cervical and first dorsal roots. This low and limited centre of origin is of importance, as it is the first to be affected by the ascent of disease from the dorsal cord, and its affection gives an indication of the exact site of the lesion. The nerve supplies the ulnar flexor of the wrist and one-half of the deep flexor of the fingers. In the palm it supplies all the muscles of the little finger, the two inner lumbricales, the interossei, the adductor, and the inner head of the short flexor of the thumb.

It supplies sensation to the ulnar side of the hand, back and front, to the palmar aspect of the fourth finger and one-half of the third, and to the dorsal aspect of the fourth, third, second, and one-half of the first, thus overlapping the area supplied by the radial. As already said, however, the median very often supplies the dorsal aspect of the terminal phalanx in the first, second, and third fingers.

Paralysis.

Etiology.—The ulnar nerve may be injured high in its course by disease of the brachial plexus, pressure of tumours, by dislocation or fracture of the humerus, or by incised or gunshot wound in the upper arm. Its exposed position at the elbow renders it liable to injury, or to pressure, and it may be implicated in disease, fracture, or dislocation of the elbow joint. It has been paralysed by pressure during sleep or over-extension during long narcosis. In the forearm it is very commonly injured in cuts, lacerations, or gunshot wounds, or in fracture of the bones of the forearm, and, like the median, may be compressed by holding an article between the arm and the side, as in some of the trades, or by the compression of a ligature during operation, or when the hands are bound behind the back. Over-exertion of the muscles it supplies, or pressure on the nerve itself, as in some occupations which involve holding an implement firmly in the hand, may injure it in the palm. Tumours are sometimes present in the course of the nerve, and cysts are found in it. It is less liable than the other nerves to be affected after the acute infective diseases, but is not uncommonly paralysed by the neuritis which appears in the puerperium. It is involved in chronic poisoning by lead or arsenic, and in some chronic spinal diseases, especially in tabes and the Duchenne-Aran type of progressive muscular atrophy.

Symptoms.—When the paralysis of the ulnar is complete, there is partial loss of the power of flexion of the wrist and fingers. The flexion is weaker than usual, and during the act there is a certain amount of inclination of the hand to the radial side, a distortion which may become permanent. The paralysis of the adductor pollicis prevents adduction of the thumb; the little finger is almost completely paralysed. The fingers cannot be flexed at the first joint nor extended at the others, from loss of power in the interossei, so that the movement of the fingers is greatly impaired, and certain actions, such as writing, are interfered with. The loss is not so evident in the first and second fingers, as the unaffected lumbricales still act. There is also loss of the power of abduction and adduction of the fingers, since these actions are normally performed by the interossei. If the nerve is affected by a lesion in the lower part of the forearm, the deep flexor of the fingers escapes, and the "claw hand" is produced. In this condition the distal phalanges are flexed by the deep flexor tendons, since extension at those points is impaired by loss of the interossei, and the proximal phalanges are extended by the extensors of the fingers which are unopposed by the paralysed interossei. The area of anæsthesia varies greatly, and is not always proportionate to the severity of the injury to the nerve.

Subjective sensations, such as tingling or numbness, are sometimes present, and may persist after the paralysis has gone. Atrophy of the muscles appears in old-standing cases, most marked in the hypothenar eminence, and the palm of the hand, which becomes hollow with marked depressions between the metacarpals. The thenar muscles stand out strongly by contrast.

Diagnosis.—The diagnosis of disease of the ulnar nerve is easy, the only condition which could cause confusion being a small lesion at the lower part of the cervical enlargement of the cord—a rare occurrence, however, as disease of this part of the cord generally spreads upward from the dorsal region, and would show other symptoms.

Prognosis and treatment.—The prognosis and treatment are the same as in the case of the musculo-spiral and other nerves of the arm.

COMBINED ARM PARALYSIS.

Some of the injuries which produce paralysis of one nerve of the arm may affect others at the same time. The most common are disease or injury of the spinal cord, the nerve roots, or the cervical vertebræ, morbid processes in the neck, dislocation of the shoulder (especially subcoracoid), fracture of the clavicle or the humerus, disease of the brachial plexus, such as neuritis, neuroma, or the ascending neuritis known as neuritis migrans, in which inflammation in a single nerve spreads upwards and attacks other trunks. A primary neuritis also occurs in the brachial plexus. The symptoms of the various isolated paralyses and the condition of the electrical reactions have already been described.

Erb's Paralysis.

Erb has described a special form of combined paralysis of the shoulder and upper arm occurring in young adults, in which the paralysis appeared in certain groups of muscles, but not confined to one nerve or always affecting the whole of any one nerve. The ulnar nerve is never affected; and, as a rule, sensation is normal, except in some branches of the median. The deltoid, supra- and infraspinatus, biceps, brachialis anticus, and supinators are the muscles affected. Since the paralysed nerves—circumflex, musculo-cutaneous, and musculo-spiral—lie far apart, the lesion must be in the cord or in the plexus before it subdivides. Erb found a spot between the scaleni, corresponding to the fifth and sixth cervical root, at which stimulation by electricity set all the above muscles in motion. Hoedemaker found this motor point in a line drawn from the sterno-clavicular articulation to the seventh cervical spine, nearly three-quarters of an inch from the anterior border of the trapezius.

In this paralysis the arm cannot be abducted nor raised to the horizontal plane, the forearm cannot be flexed on the arm, and there are subjective sensations of tingling and numbness on the radial side of the forearm, the back of the thumb, sometimes also in the ball of the thumb, and the tips of the first two or three fingers. The ulnar nerve and the other branches of the median are normal. The atrophy of the muscles may become very marked, and the electrical reactions are altered, the reaction of degeneration being present. Duchenne and Seligmüller describe a similar paralysis which occurs in infants as the result of the pressure on the arm, or traction on the neck by the hook or the fingers during delivery.

The prognosis in the case of adults is unfavourable. Recovery is slow, or the condition remains permanent. In the obstetrical cases it is more favourable, recovery being slow but perfect.

The treatment of this condition is similar to that of the other paralyses of the nerves of the arm.

KLUMPKE'S PARALYSIS.

There has recently been described a form of paralysis due to injury of the lowest roots of the brachial plexus, which, although recognised for some time, had not been fully investigated before. It is characterised by paralysis and atrophy of the muscles of the thenar and hypothenar eminences, and of the interossei. Sensibility is diminished in the areas supplied by the ulnar and median nerves, and there is some disturbance of the cilio-spinal centre. The palpebral fissure on the affected side is narrowed, the eyeball sunken, and the pupil contracted. The condition is produced by injury to the plexus in forcible reduction of a dislocated shoulder, from the extirpation of a tumour, or the pressure of a tumour of the lungs or vertebral column. Some cases have been described in which the three forms of paralysis (Duchenne, Erb, and Klumpke) were combined.

BRACHIAL NEURITIS.

The brachial plexus, like the sciatic nerve, is subject to an excessively painful form of spontaneous neuritis in which one or more branches or the whole of the plexus may be involved. The disease is a perineuritis, an inflammation of the sheaths surrounding the nerve fibres, and is apparently due to gout, very often the inherited form.

This neuritis of the brachial plexus is found in women much more frequently than any other form, the sexes being about equally affected. It is usually preceded in men by typical gouty attacks, and in women by muscular rheumatism, most commonly lumbago, and by sciatica. It is a disease of the later period of life, very few cases occurring before fifty, and many of them persisting to very advanced life.

Symptoms.—Pain is the most prominent symptom, and is even worse in this form of neuritis than in other varieties. It precedes the inflammation, often persists long after the disease is gone, and its severity, occurring at such an advanced age, may render it very serious. The pain is very often referred to points at some distance from the actual lesion, to the scapula or wrist, but more often is present above the scapula or in the axilla, from whence it extends along the lines of the nerves. It is usually slight at first and intermittent, but becomes more continuous afterwards, with paroxysmal exacerbations. In the intervals the pain is dull and gnawing, and in the paroxysms may be acute, stabbing, burning, or lancinating, darting along the nerves and extending to the side of the thorax and neck. Movement of the arm, especially elevation, greatly increases the pain. In slight cases it may bring on an attack, and severe cases are always aggravated by it, the consequence being that the limb is kept as still as possible to avoid the suffering. The skin is often hypersensitive during and after the attacks, but loss of sensation seldom occurs except in long-continued cases. When both pain and loss of sensation are present, the condition is known as anæsthesia dolorosa.

The muscles are not, as a rule, much atrophied or altered in their electrical reactions, except in some cases where the inflammation attacks a certain portion of the plexus so severely as to seriously damage the nerve fibres. They are very flabby and weak, but the flabbiness and atrophy are due to the forced inaction, and the weakness, to a great extent, to the dread of movement. The skin may become thin and glossy, with some subcutaneous œdema, and arthritic changes are almost invariable when the disease occurs in old age, often leaving permanent adhesions within the points.

Diagnosis.—The diagnosis of brachial neuritis is not always easy, the conditions with which it is likely to be confused being neuralgia in the nerves of the plexus and angina pectoris. Both neuralgia and neuritis may be due to gout, so that there is little help to be gained from the history except as to the onset. The situation of the pain, the tenderness over the nerve, the increase of pain on movement, and evidence of degeneration of the nerve fibres, would all suggest neuritis, but it may be very difficult to decide.

Confusion with angina pectoris is likely to arise in cases where the inflammation is situated high in the plexus, giving rise to pain at remote parts and to irritative symptoms, especially if some condition of the heart be present which would render angina probable. The pain in such cases often resembles the pain of angina very closely. It occurs in paroxysms, radiates down the arm and to the side of the chest, and, like all pains in the left brachial region, has a tendency to affect the action of the heart, and to suggest cardiac disease or aneurysm. Tenderness along the line of the nerves is nearly always present in neuritis; in the case of aneurysm or cardiac disease the pain is more intense, constant, and progressive, while tenderness over the nerves is absent, and other symptoms of the lesion are usually present. The possibility of the presence of acquired or inherited gout should be borne in mind. In chronic stages the neuritis may simulate chronic primary arthritis with consequent atrophy, but the character and development of the disease are usually distinctive.

Prognosis.—The prognosis as to ultimate recovery is fairly good; but the disease may persist for months, a year, or even longer if the patient is aged, and in all cases relapses are common, more so after a slight than after a severe attack. Unfortunately, the recovery is not always perfect. The limb may be left weak and small, and cramp, neuralgia, or tingling may occur in it. The joints may be considerably altered, since adhesions may form, owing to the fixation of the joint and the impairment of the nutrition of its tissues. These changes are especially common in the shoulders, the wrists, and the fingers. The fingers are often very painful, from the strain on the long-unused joints, when an attempt is made to use them after the neuritis has subsided. It must be remembered that the gouty tendency of the patient may aggravate the morbid condition of the joints.

Treatment.—The treatment is the same as that already given for other forms of neuritis, but absolute rest and immobility are even more imperative here than in other cases, even at the risk of fixation of the joints. The most useful drug is cocaine, administered subcutaneously twice a day in doses of one-tenth to one-third of a grain. Morphine may also be injected. In the early stages neither massage nor electricity should be used, but when the pain and tenderness are past, the limb may be rubbed very lightly. Late in the disease, if reaction of degeneration has appeared in the muscles, electricity may be employed.

THE ANTERIOR CRURAL NERVE.

The anterior crural nerve rises chiefly from the third and fourth lumbar nerves, but partly also from the first and second. It supplies motor fibres to the psoas and iliacus, the quadriceps extensor, the sartorius, the obturator, and the pectineus. Its sensory branches are the anterior and middle cutaneous and the long saphenous nerves, which supply the whole of the thigh, except a narrow strip up the back, the inner aspect of the leg and foot, and the adjacent sides of the first and second toes.

The anterior crural nerve may be injured directly by a stab or shot in the thigh, by fracture or dislocation of the femur, the pelvis, or the lumbar vertebræ. Disease, especially inflammation, of the bones of the pelvis, or the cellular tissue within it, of the femur, or of the ilio-psoas muscle, may cause lesions of isolated branches of the nerve, as also may over-exertion of the muscles. The nerves of the lower limb, the anterior crural especially, are very often involved in degenerative neuritis, such as occurs in chronic alcoholism, after acute infective diseases, and in glycosuria. The anterior crural nerve is involved in many of the central diseases, especially lesions of the cord, such as muscular atrophy and atrophic spinal paralysis in children, etc.

When the whole nerve is paralysed, flexion at the hip and extension at the knee are lost, so that standing and walking, and more especially climbing, jumping, or running, are very difficult, or, when the lesion is bilateral, impossible. When the lesion is unilateral, the patient can stand alone, and may be able to walk by dragging the paralysed limb after him. When the disease is within the abdomen, the psoas may escape, so that flexion at the hip is weakened but not lost. The amount of paralysis varies with the site and character of the lesion. The knee-jerk is lost. Anæsthesia is usually present in the whole area supplied by the nerve, and the irritation of the nerve may cause great pain in the same area. This is sometimes the first symptom of a growth near the spine. Pain may pass along the upper branches of the plexus (the ilio-hypogastric, the ilio-inguinal, and the genito-crural) when the whole plexus is involved in a neuritis.

THE OBTURATOR NERVE.

The obturator nerve rises from the second, third, and fourth lumbar nerves, and is distributed to the adductors, the gracilis, the obturator externus, and part of the pectineus; it also gives branches to the hip and knee joint. One of its branches anastomoses with the internal cutaneous nerve, and supplies the inner side of the thigh as far down as the knee.

Isolated paralysis of the nerve is rare. It is sometimes damaged within the pelvis during difficult labours, especially when delivery is instrumental, or it may be compressed by an obturator hernia. More commonly the anterior crural nerve is also injured.

Paralysis of the obturator nerve causes loss of adduction of the thighs, and of the power of crossing the legs over each other. External rotation is diminished by paralysis of the obturator externus, and internal rotation by the paralysis of the lower fibres of the adductor magnus. The power of grasping the saddle firmly with the thighs in riding is also lost. There is anæsthesia of the inner side of the thigh and knee, and in cases of obturator hernia a burning pain may be felt in this region.

THE SACRAL PLEXUS.

The branches of the sacral plexus give off four large nerves, the superior and inferior gluteal, and the small and great sciatic.

The three first nerves may be injured by direct injury to the sacrum or pelvis, such as fracture, or the severe contusion and hæmorrhage which occur after a fall in a sitting position. Tumours of the sacrum, pelvis, or cauda equina may also injure these nerves. The glutei are involved in the muscular atrophy which occurs in pseudo-hypertrophic paralysis.

The superior gluteal nerve rises from the lumbo-sacral cord, and supplies the gluteus medius and minimus and the pyriformis, so that when it is paralysed there is loss of power of abduction and circumduction of the thigh.

The inferior gluteal nerve supplies the gluteus maximus, which is the extensor of the hip-joint, acting as antagonist to the ilio-psoas. Paralysis of the inferior gluteal will therefore cause loss of extension at the hip-joint, and of movement of the limb backwards beyond the vertical line.

The small sciatic nerve is purely sensory in its function, and is distributed to the gluteal region, to the inner, upper portion of the thigh, and to its posterior aspect as low as the popliteal space and the upper part of the back of the leg. It also supplies a small branch to the perinæum. It is very seldom paralysed alone, but frequently along with other nerves. The result of its paralysis is anæsthesia in the area it supplies.

THE SCIATIC NERVE.

The great sciatic nerve arises from the fifth lumbar and the first, second, and third sacral nerve roots. It supplies motor fibres to the external rotators of the thigh and to the hamstring muscles (the biceps, semi-tendinosus, and semi-membranosus), and gives a small branch to the adductor magnus. By means of its two great terminal branches, the internal and external popliteal, it supplies all the muscles of the leg, and it is the sensory nerve to the leg and foot, except their inner aspect, which is supplied by the internal saphenous.

The great sciatic is more frequently affected by injury or disease than any other nerve of the leg, because of its long course and exposed position. It may be injured by fractures of the sacrum or pelvis, by concussion or hæmorrhage within the cauda equina from a severe fall, by fracture or dislocations of the femur, by surgical operations in its neighbourhood, or by incised or gunshot wounds in the thigh. During difficult labours it may be compressed by the head of the child. It is very frequently affected by degenerative neuritis after acute infective diseases, and it is frequently the seat of a spontaneous neuritis (sciatica), probably due to inherited gout or rheumatism.

When the whole nerve is paralysed, there is great impairment of the power of external rotation of the thigh, and of extension at the hip-joint, complete loss of the power of flexion at the knee-joint, and complete paralysis of the leg and foot. Walking, climbing, running are thus rendered very difficult, and the leg can only be used in rigid extension. There is anaesthesia of the outer half of the leg, the dorsum and sole of the foot, and the toes. The sensibility of the leg is sometimes unaffected, since there is a free communication with other nerves in this situation.

THE EXTERNAL POPLITEAL NERVE.

The external popliteal nerve through its branches supplies the tibialis anticus, the peronei, the long and short common extensors of the toes, and the extensor of the great toe. The exposed course and the close proximity of the external popliteal nerve to the bone render it very liable to direct injury, and it is sometimes the seat of spontaneous neuritis. When it is paralysed, the foot cannot be dorsiflexed, and the toes cannot be extended at their proximal phalanges. Abduction and adduction are greatly impaired, and the patient walks in a characteristic manner, with the toe pointed and tending to trail along the ground, so that the knee-joint must be flexed in an exaggerated degree to bring the foot forward (*steppage*). The contracture of the plantar muscles produces a condition of talipes equinus in long-standing cases. Anæsthesia is present in the outer half of the front of the leg, the outer portion of the calf, the dorsum of the foot, and the dorsal aspect of the toes, areas which receive their sensory supply from the terminal branches of the external popliteal, namely, the musculo-cutaneous and the anterior tibial nerves.

THE INTERNAL POPLITEAL NERVE.

The internal popliteal nerve supplies the flexors of the leg, ankle, and toes, giving motor fibres to the popliteus, the gastrocnemius, tibialis posticus, long flexors of the toes, and the short muscles of the sole of the foot. It gives sensory fibres to the lower part of the calf, the outer margin and sole of the foot, and the plantar aspect of the toes.

When the internal popliteal nerve is paralysed, the power of extension of the ankle joint is lost, the patient cannot rise upon his toes, and talipes calcaneus is produced. If the injury is above the popliteus muscle, there is loss of inward rotation of the leg in addition. Anæsthesia is present in the area supplied by the nerve.

The plantar nerves are rarely paralysed alone, but a lesion may injure one or other in the sole or ankle. When the internal plantar is affected, the short flexors of the toes, the plantar muscles of the great toe (except the adductor), and the two inner lumbricales are paralysed. There is anæsthesia over the inner part of the sole, the plantar aspect of the three inner toes, and one-half of the fourth toe. When the external plantar is affected, there is paralysis of the flexor accessorius, all the interossei, the two outer lumbricales, and the adductor of the great toe. The toes become flexed at the last two phalanges and extended at the first (a position analogous to the "claw-hand"), and the loss of the power of propulsion of the body in walking causes serious inconvenience. Anæsthesia is present in the outer half of the sole, the plantar aspect of the little toe, and one-half of the fourth toe.

Diagnosis.—The diagnosis of a nerve affection in paralysis of the muscles of the lower extremity is not difficult when there is an obvious lesion in the pelvis, or in the limb itself, implicating the nerve. A knowledge of the function of the nerves, and of the abnormalities in position, movement, and sensation produced by their paralysis, will determine which nerves are affected, and the degree of injury. Atrophy of the muscles, loss of reflexes, and changes in electrical reaction would also serve to indicate the affected nerve.

It is sometimes exceedingly difficult, however, to distinguish between peripheral and central paralysis. The relation of the nerve trunks to the roots is simple, so that a limited lesion of the cord may affect the function of one nerve only, most frequently the anterior crural and external popliteal, and thus simulate a peripheral lesion of the nerve. A small lesion in the course of the cauda equina may also affect a limited number of fibres, and thus suggest a lesion in a single nerve.

A careful examination of the patient will usually show other symptoms, such as implication of the pelvic viscera, if the cord is affected, and paralyses due to involvement of the cord or cauda equina are usually bilateral, unless in the case of a limited lesion in the anterior horn of the cord. When the lesion is in the white matter of the cord, the reflexes are exaggerated, atrophy does not follow, and sensation is often unimpaired; whereas in injuries to the nerve the reflexes are abolished, atrophy sets in rapidly, sensation is lost, and reaction of degeneration soon appears. The history is of great use, as a recent difficult labour, puerperal fever, acute infective disease, poisoning by alcohol or lead, would indicate a neuritis; whereas the typical history of acute anterior polio-myelitis in children, tabes in adults, injury or dislocation of the lumbar vertebræ or sacrum, would indicate a lesion in the cord.

Prognosis.—The prognosis in cases of paralysis of the nerves of the leg depends upon the character and site of the lesion, and, speaking generally, is the same as already given in other cases of peripheral affections of the nerves. The electrical reactions are important. The cases in which they are only slightly altered recover rapidly, whereas when reaction of degeneration has appeared, either recovery is slow or the paralysis is permanent. The paralysis which results from disease of the cord is much more serious than that which follows a lesion of the nerve, and the prognosis is absolutely unfavourable in the progressive muscular atrophy and pseudo-hypertrophic paralysis of children.

Treatment.—This is similar to that which has already been described in full in dealing with paralysis in the arm, namely, sedatives and rest in the early stages, and electricity and massage in later stages. In traumatic cases, or in the case of pressure from a tumour, surgical interference will be required, and when the paralysis is due to poisoning by alcohol, lead, or in diabetes, suitable treatment must be employed. In the case of alcoholism, total abstinence from any form of alcohol is imperative. The pain, which may be very acute, is best treated by antipyrine, antifebrin, or, as a last resource, opium. Attention to the position of the limb is even more necessary in the legs than in the arms, since contraction of the hamstrings or of the calf muscles may cause the utmost inconvenience. When the calf muscles are paralysed, it may be possible to prevent over-extension of the foot by the use of sandbags, or a board across the bed to keep the foot at right angles to the leg.

SCIATICA.

The term sciatica, as commonly used, simply means pain along the sciatic nerve, irrespective of the cause. It should properly be limited to the pain produced by neuritis, or neuralgia of the nerve, independent of any external cause, and will be used in that sense here.

Etiology and pathology.—Sciatica is much more common in men than in women, the proportion being about four to one. It occurs most frequently between 30 and 60, the maximum being reached between 40 and 50. It never occurs in childhood. There is little doubt that gout and rheumatism are the chief predisposing causes. Many patients show evidences of a gouty state, such as tophi, Heberden's nodules, or have been subject to previous attacks of muscular rheumatism.

The immediate exciting cause is usually exposure to cold and wet, either local, such as wetting the feet or sitting on wet grass, or a general chill. The nerve may be injured by external pressure, usually that of a hard seat when the occupation is sedentary, or by over-exertion of the muscles. Inflammation may extend from surrounding disease to the nerve, as in rheumatic inflammation of adjacent fibrous tissue, such as lumbago, or inflammation in the pelvic cellular tissue. Pressure on the nerve by a tumour or by the fœtal head during labour may excite a neuritis. Disease of bone outside the pelvis, especially the hip-joint, may also extend to the nerve.

Symptoms.—Pain is the most prominent symptom in sciatica, as in other forms of neuritis. In a typical case there is generally some slight prodromal pain for a few days, which afterwards becomes more marked in any posture which puts a strain upon the nerve, or causes pressure upon it. The pain is increased by movement, so that the knee is kept slightly flexed in walking, and the patient walks upon his toes with the leg kept as rigid as possible. It may increase to such a degree, that perfect rest in a position in which the nerve is completely free from tension is the only condition possible for the patient. In the acute stages of the disease, spontaneous pains radiating along the nerves appear in addition to those induced by movement. The pain is felt most acutely at certain spots, namely, above the hip-joint; near the posterior iliac spine; at the sciatic notch; about the middle of the thigh; behind the knee; below the head of the fibula; behind the external malleolus; on the back of the foot.

The character of the pain varies. It is seldom purely paroxysmal, but is more often steady and dull, or acute, with exacerbations. It is often burning, radiates from above downwards, and may sometimes extend to all the branches, marking out the course of the nerves. Tenderness on pressure over the nerves becomes very marked as the disease progresses, especially along the back of the thigh. Pressure on the external popliteal nerve where it lies close to the tendon of the biceps is excessively painful.

Paræsthesiæ are common, tingling, numbness being present, and there are irregular patches of anæsthesia in the areas supplied by the nerve. The back of the thigh may be anæsthetic, showing that the small sciatic is involved. Slight wasting of the muscles of the hip, thigh, and leg, and flabbiness due to disuse, are usually present, but it is only in severe cases that these changes are extensive, or that the electrical reactions are markedly altered. There is sometimes evidence of vasomotor changes, the limb being paler and colder than its fellow. Cramps and fibrillar contractions sometimes appear in late cases. Sciatica is almost never bilateral, but has been known to ascend to the cord and pass thence to the nerve on the opposite side. It has also been known to extend to the lumbar plexus, and involve the nerves on the front of the thigh.

Diagnosis.—The typical position of the pain, its relation to the nerve and its branches, and the tenderness on pressure, serve to distinguish true sciatica. Whether it is primary or secondary, will be decided by careful examination of the sacrum, the pelvis, the hip-joint, for cellulitis, tumour, or disease of the bone.

In neuralgia the subjects are generally ill-nourished and anæmic, the pain is entirely spontaneous, and is not influenced by posture or movement, there is little or no tenderness on pressure, and the pain is felt in the branches rather than in the trunk of the nerve. The conditions with which it is likely to be confused are neuralgia of the nerve, lumbago, hip-joint disease, sacro-iliac disease, and lesions in the vertebræ, the cauda equina, or the cord itself. Lumbago can usually be easily distinguished by the situation and limitation of the pain. Disease of the hip-joint or sacro-iliac joint will be distinguished by other symptoms of such an affection, and a proper examination of the movements, etc., will easily reveal the condition. In cases where the spine, the cauda equina, or the cord are the seat of the lesion, there is usually no tenderness over the nerve, the pain is peripheral in its distribution, and in the last two conditions the pain is almost always bilateral, whereas true sciatica is rarely present on both sides.

Prognosis.—In slight cases the prognosis is favourable, recovery usually taking place within a few weeks. It is, unfortunately, much more common for it to take many months, or even a year, to subside, during which time the spontaneous pain may be continuous and intense. Relapse and recurrence are both common. The lessening of spontaneous pain is the first sign of recovery, and is followed by the gradual diminution of pain on movement. The wasting, fibrillary tremors and cramps may persist for months, or even years, after the attack is over.

Treatment.—In a case of sciatic pain the pelvis and the pelvic organs should be carefully examined to exclude the possibility of tumour, aneurysm, or disease of the bones; when present, these must of course be treated according to their nature.

Gouty cases should be treated with salines, and salicylates may be tried where rheumatism is suspected to be the-cause. When an attack has begun, absolute rest in a posture that relieves the nerve of any tension is imperative. Fixation by a long splint is often of use. Hot poultices may be applied in the line of the nerve, or counter-irritants, such as mustard blisters or the thermo-cautery. Sometimes sedatives may be rubbed in, such as liniment of belladonna or aconite. Internally the drug that is of most use is mercury—one grain of blue pill twice daily when acute inflammation is thought to be present. Quinine, antipyrine, and antifebrin are of little use, but the use of cocaine or morphine subcutaneously is very successful. Cocaine, injected near, but not into, the nerve in doses of one-eighth to one-half grain twice daily, sometimes does well. Chloroform (15 to 30 minims) injected into the nerve has answered in some cases, as also injections of distilled water. Morphine injections (one-twelfth to one-sixth grain) should only be tried as a last resource, the temptation to the formation of habit is so great when the patient is worn out with continuous pain.

Acupuncture along the nerve has sometimes proved of great use, the needles being thrust deeply into the painful part, and left in place for about twenty minutes. Nerve-stretching has also been tried, but the results are not certain. Electricity is of little use, except in the later stages. After an attack the patient's general health should be watched, and a threatening attack treated promptly by rest and counter-irritation.

MULTIPLE NEURITIS.

Multiple or polyneuritis is the term applied to the condition in which many of the nerves are inflamed, usually symmetrically, and sometimes successively, by the presence of some infective or toxic substance within the body. It has also been called peripheral neuritis, owing to the fact that the nerves are first and most severely affected at their peripheral terminations.

Etiology.—The condition is due, as already said, to the presence of some injurious substance. This may be—

Toxic, such as alcohol, lead, mercury, arsenic, carbon—bisulphide of naphtha. The toxic agent in diabetes and rheumatism may also be included here.

Infective, diphtheria (influenza), malaria, typhoid and scarlet fevers, leprosy, smallpox, syphilis, tuberculosis, beri-beri.

Cachectic and senile, cancer, anæmia.

It is a disease of adult life, most of the cases occurring between the ages of 20 and 50. In children it is very rare, and only appears associated with polio-myelitis or as multiple adventitious neuritis. The female sex is more often affected than the male, owing to the greater frequency of the alcoholic form in women. The rheumatic and toxæmic forms are more common in men. The immediate cause may be physical depression, due to chill or exposure, or to mental or emotional disturbance.

Symptoms.—The symptoms in multiple neuritis vary greatly, as has been said, according to the cause and extent of the lesion. They are mainly motor and sensory, but some cases occur in which the motor and sensory symptoms are inconspicuous, and the prominent feature is marked inco-ordination of the muscles which gives rise to an ataxic condition.

As alcoholic neuritis is the most common and most important form of the disease, it may be taken here as the type. The condition may appear in an acute, a subchronic, or a chronic form, the subchronic being most common, and therefore described first. In cases of subchronic alcoholic neuritis there are usually some premonitory symptoms, such as numbness, tingling, cramps, and dull rheumatoid pain in the extremities, symptoms which may remain stationary until some impairment of vitality aggravates the disease, when they become more marked, and are accompanied by more severe pain, impairment of motion, paralysis or inco-ordination.

The motor paralysis is usually symmetrical, attacking both hands and feet, but may be confined to, or more severe in, upper or lower extremities, according to the cause of the neuritis. In all cases the extensors are more affected than the flexors, so that wrist-drop and foot-drop ensue. The paralysis may be limited to the extremities, or may go on to involve the muscles of the thigh and upper arm, the face, the sphincters, the larynx, and the respiratory muscles. The effect of the paralysis of the flexors of the foot is to produce a curious high-stepping gait, "steppage," in which the knee is very much flexed and raised in order to bring forward the toes, which remain pointed and drooping. When inco-ordination is the main symptom, the gait may be more distinctly ataxic. In the leg the muscles first and most severely affected are those supplied by the external popliteal nerve (namely, the tibialis anticus, peronei, and extensors). In the arm the muscles supplied by the posterior interosseous nerve (from the musculo-spiral) are the first to be attacked as a rule. In the neuritis due to lead-poisoning the supinator longus and the long extensors of the thumb escape.

In severe cases the muscles of the thorax and abdomen are involved, and sometimes, though very rarely, the larynx and the heart are affected by paralysis of the vagus nerve. The facial and ocular muscles escape, except in exceedingly severe cases, and the pharynx and alimentary canal are never involved. Sensation may be unaffected, or its disturbances may be slight or marked. When affected, tingling, numbness, or hyperæsthesia may be present. The sensibility of the skin to touch and temperature, especially in the distal parts of the limbs, is usually lessened early and persistently, while sensibility to pain, though often delayed and slow, may be excessive when once elicited.

Spontaneous pains accompany the hyperæsthesia, and there is tenderness over the affected nerve trunks. The muscles are also excessively tender on pressure. These abnormalities of sensation are most marked in the extremities, especially on the palms and soles, and on the finger-tips, but may become much more widely spread as the motor system extends.

The conditions of the superficial reflexes vary. If there is much hyperæsthesia they may persist even when the motor symptoms are marked, but they disappear at an early stage if the cutaneous sensibility is lowered. The tendon reflexes are lost in nearly every case, but may be excessive in the very early stages, probably from the irritability of the nerves. The organic reflexes may be affected in severe cases, the urine and fæces being passed involuntarily. The affected muscles rapidly atrophy and become soft and flabby, the atrophy progressing to a high degree. Their electrical excitability alters, the reaction to faradism being diminished or lost, while the response to voltaism is excessive and usually altered in quality. Both faradism and voltaism may fail to produce a response in the nerves in late cases.

The other trophic changes are similar to those in other forms of neuritis, the most common being glossy skin and adhesions within the joints. Œdema is common, especially over the joints, and there may be pain and effusion into some of them, most commonly the fingers or wrists, followed by adhesions, which may be permanent and cripple the patient. Once formed, the adhesions are apt to reappear after being broken down. Bedsores rarely form.

The symptoms vary somewhat according to the nature of the exciting cause. In alcoholism all the limbs are affected, but the legs suffer first and most severely. The metallic poisons usually affect only the arms, and malarial poison only the legs. The neuritis is most widespread in toxæmic conditions, in cold (rheumatic neuritis), and in alcoholism especially, when the laryngeal, cardiac, and respiratory muscles may be affected. The face, however, is never affected in alcoholism, and the larynx is almost invariably spared in all cases. The optic nerve is only rarely affected.

The acute form of multiple neuritis comes on suddenly after exposure to cold or to some toxic agent. It may set in with a rigor, some pain in the back, and a rapid rise of temperature, which may reach 103°–104° F. The pain in the muscles is not great, numbness and tingling being the main symptoms, but motor paralysis sets in early and advances rapidly. Death may result within a fortnight, or even a week, from paralysis of the respiratory muscles.

The course of an ordinary case of multiple neuritis varies with the intensity of the attack, and the degree in which it can be controlled and its cause suppressed. Under treatment, neuritis usually increases for three to six weeks, remains stationary for a time, and then slowly recovers. Untreated, the disease is steadily progressive. The power returns first to the muscles that have been least affected, but is slow in coming back to those that have been much atrophied. The contracture of the opposing muscles also disappears gradually. As already said, it is most serious in the calf muscles, as it renders walking impossible. The first sign of recovery is the diminution of spontaneous pain and hyperæsthesia, which disappear long before the pain on movement, and the tenderness on pressure. Relapses are very common if the patient's vitality is lowered, or if he is again exposed to the poison.

A fatal termination in multiple neuritis is very often due to paralysis of the respiratory muscles, but may be due to some complication, such as heart failure or exhaustion, in long-continued cases. Some pulmonary affection, pneumonia, phthisis, or bronchitis, may supervene and prove fatal. In alcoholic subjects, cirrhosis of the liver, gastritis, or cerebral meningitis are common, and in diabetes, acetonæmia may prove fatal.

Diagnosis.—The diagnosis of a typical case of multiple neuritis is not difficult. Its localisation, symmetrical distribution, onset, and the tenderness of muscles and nerves, taken together with the history of a distinct cause, serve to distinguish it from other paralytic conditions. The diseases with which it is likely to be confounded are anterior polio-myelitis, tabes, Landry's ascending paralysis, and hysterical paralysis.

From polio-myelitis it is distinguished by its symmetry, the affections of the muscles in polio-myelitis being quite unsymmetrical, the persistent pains, the tenderness over the nerves, and the sensory changes. From tabes it may be distinguished by the gait, which differs from that of ataxia, the absence of pupillary changes, girdle or lightning pains, visceral disorders or crises, or paralysis of the sphincters. The inco-ordination is not so marked in neuritis, the patient being usually able to stand alone with the eyes shut, and there is evident wasting and flabbiness of the muscles. Some observers consider that acute multiple neuritis and Landry's ascending paralysis are the same disease, but in neuritis the paralysis attacks both extremities at once, whereas Landry's paralysis begins in the legs, passes to the trunk, and invades the arms last. In hysterical paralysis the extensors are never affected in the manner characteristic of neuritis. The knee-jerk is not lost, and when anæsthesia is present it is much wider in extent than in multiple neuritis.

Prognosis.—In cases of acute neuritis due to cold or some toxæmic state, the prognosis is very grave, as in these cases there is a great tendency to rapid extension of the paralysis to the muscles of respiration. In the subacute cases also, the prognosis is grave if the patient is very weak, or if the paralysis has involved all the limbs and some of the muscles of the trunk. Since the disease tends to progress for two or three weeks after the cause has been withdrawn, there is a risk that in such advanced cases the respiratory muscles may be affected before the stationary period arrives. Complications, such as bronchitis, pneumonia, meningitis, nephritis, hyperpyrexia, also tend to render the condition a grave one. In favourable cases of subacute and chronic neuritis, recovery usually takes place within six months.

On the other hand, slight cases recover rapidly and completely under treatment if begun early. The prognosis as to duration of the paralysis depends upon the condition of the muscles. When these are highly atrophied, it may be months, or years even, before they recover, but the recovery is generally permanent and complete. The sensory symptoms disappear along with the motor, the tenderness of the nerves and muscles being last to go. The prognosis as to the time of recovery of power in the muscles can be given most certainly by the aid of electricity. If the reactions are much altered, especially if complete reaction of degeneration be present, recovery will be slow. Return of faradic irritability is a sure sign of improvement in the muscles.

Treatment.—Rest in bed is essential, and, where possible, the cause of the neuritis must be removed. In alcoholic cases especially it is absolutely necessary that no alcohol should be taken, unless great prostration or cardiac weakness should indicate otherwise. In many cases, especially in private practice, it is not easy to make sure that the supply of alcohol is quite cut off, as alcoholic patients become exceedingly cunning and unscrupulous, and will make any effort to obtain their stimulant. If there is a relapse or absence of improvement in such a case, it is very probable that alcohol is still being obtained.

Careful feeding is of the greatest value in treating neuritis. The food should be nourishing and easily assimilable, and given at regular intervals of not more than three hours. Milk, cream, custard, and beef-juice are all valuable in the more acute stages, supplemented, as recovery sets in, by more solid food.

Hot fomentations over the painful muscles give great relief; but care must be taken that they are not too hot, as vesication is easily produced. Counter-irritation may also be employed, but, unfortunately, neither treatment has much effect on parenchymatous neuritis. Many drugs have been employed to relieve the pain, the most useful being cocaine, which may be injected subcutaneously in doses of one-twelfth to one quarter of a grain twice daily. Besides deadening pain, it has the further advantage of lessening the craving for alcohol. Morphine should not be used except as a last resource, especially in alcoholic cases, as the opium habit is very readily formed. To lessen this risk, the opium should, if possible, be given by the mouth; and if given subcutaneously, the injections should be given by the physician himself, or, in extreme cases, by a skilled attendant; but in no case should they ever be entrusted to the patient.

Phenazone and acetanilide are useful in minor cases, but their tendency to produce anæmia is a drawback to their continuous use. Cannabis indica may be given steadily, and in combination with other drugs. Bromides, sulphonal, trional, and chloralamide may be used to overcome the sleeplessness, and soothe mental excitement.

No drug has any specific influence on the general course of the disease, but an acute attack may be treated by diaphoretics; and when rheumatism is suspected, salicylate of soda may be employed. In chronic cases, strychnine, iron, and quinine may be given for their tonic effects, and iodide of potassium is of use in the chronic sensory form. Arsenic may also be tried in chronic cases, but its action must be carefully watched, as it has itself a tendency to produce neuritis. Cod-liver oil may be given in late stages to improve nutrition.

Massage should be begun early, as soon as the pain has sufficiently subsided to allow the patient to bear the rubbing, in order to maintain the nutrition of the muscle and improve metabolism generally. It should be kept up throughout the course of the disease, until recovery is complete.

Electricity should also be begun as soon as the pain permits, but it is not of so much use as massage. It should be employed daily for ten to fifteen minutes in the form of the voltaic current, large sponges being used in order to cover as large an area as possible.

It must always be borne in mind that contractures form very readily in the unopposed flexor muscles, and that, therefore, the posture of the patient should be attended to. A sandbag or upright board across the bed against the feet will prevent foot-drop and contraction of the tendo Achillis and flexors of the toes; and the legs and thighs should be kept extended to control the hamstrings and gastrocnemius. The contractures, when formed, usually yield readily to massage and exercise: but in some very rare cases it may be necessary to divide the tendo Achillis.

ENDEMIC NEURITIS.

Beri-beri, or Kak-ké, as it is called in Japan, is an organismal disease prevalent in the East. It is endemic in various parts of India, China, Japan, the Eastern Archipelago, Ceylon, New Zealand, the South Pacific Islands, and the coast of Brazil. It may occur in epidemic form, but is usually confined to certain closely-inhabited places, such as barracks or prisons, where there is opportunity for repeated infection by the germs in the soil or the air.

Etiology.—The etiology of the disease is not clear. It has been attributed to various articles of food, fish, or rice, or to a deficiency of the nitrogenous part of the diet. An organism has been obtained from the blood of patients, and consists of rods and cocci. It thrives best in the blood at a high temperature, and in moist surroundings, so that the danger of infection is considerably lessened by free ventilation and precautions against damp. This fact probably explains the apparent immunity of Europeans resident in a beri-beri district.

Symptoms.—The symptoms of the disease are evidently due to a general or localised neuritis, affecting few or many nerves, and showing many variations in its manifestations. The most invariable are weakness in the legs, numbness, tingling, and œdema over the tibiæ, and extreme sensitiveness to pressure in the calf muscles. The paresis, anæsthesia, and œdema may be limited to the legs, or may become general ("wet form"), or affect separate areas, or there may be paresis and anæsthesia, associated with atrophy, but with no œdema ("dry form"). The vagus and the phrenic are frequently affected; and one of the most distressing features of the disease is the liability to sudden death from cardiac or respiratory failure in an apparently mild case. When affected in a slighter degree, there is breathlessness on exertion.

The disease may be acute or chronic. In the acute form, the neuritis is not so evident, fever, anæmia, and general dropsy being the main features; but the chronic form closely resembles multiple neuritis. There is paralysis, especially in the flexors of the ankle, atrophy of the muscles, sensory and trophic disturbances, and a varying amount of anasarca. The course of the disease varies greatly, with many exacerbations and relapses, different parts being affected. The mortality ranges from 4 to 50 per cent., and the fatal issue is usually due to cardiac failure, but sometimes to exhaustion in protracted cases.

Treatment.—The most important part of the treatment is thorough disinfection and ventilation of the dwellings, and prevention of damp or overcrowding. No preventive inoculation is as yet known; but when once the disease has declared itself, the patient should be removed from the infected neighbourhood, as there is no doubt that many protracted cases are due to repeated re-infection, and that the disease will die out if such infection be prevented. Otherwise the treatment is the same as for neuritis. Stimulating nitrogenous diet should be given, and all severe exertion avoided. In severe cases absolute rest is imperative. Digitalis and strophanthus may be given in small doses to strengthen the heart, and iron, strychnine, and quinine for their tonic action. Sudden cardiac embarrassment may be relieved by venesection.

NEUROMA.

The term neuroma was formerly applied to any new growth in connection with a nerve, but more recently investigation has shown that such new growths are only in a few cases composed of true nervous elements, whereas in the majority of cases they consist of non-nervous tissues, most commonly fibrous tissue.

Etiology and pathology.—The cause of neuroma is not known. In some cases it is hereditary, and is evidence of a neurotic disposition, occurring, as it sometimes does, associated with cretinism and imbecility. The sexes are equally affected, except in the case of multiple neuroma, which rarely occurs in women. The only immediate cause known is injury, such as pressure upon, or division of the nerves.

These tumours have been divided into true neuromata and false neuromata, according to their composition. Simple hypertrophy of nerves, an exceedingly rare condition, and the enlargement of the nerves by connective tissue which occurs in anæsthetic leprosy, do not come under consideration here.

The true neuroma contains nerve fibres medullated or non-medullated, or both, and in some few cases ganglion cells are also present. These tumours are very rare, and are found almost exclusively on spinal nerves. The nerve fibres, however, are not in connection with those of the nerve trunk, but are irregular groups of fibres separated by connective tissue.

The false neuromata vary greatly in structure, the commonest variety being the fibroma, while myxoma, sarcoma, and carcinoma of the nerves are much rarer forms, and usually affect the nerve by invading it from adjacent tumours.

There are some varieties of neuroma which have received distinctive names. The form which is most commonly found is the multiple neuroma or neuro-fibroma. As the name indicates, these tumours occur in great numbers—sometimes in thousands—throughout the body. They are subcutaneous, fusiform swellings, painless, as a rule, and vary greatly in size, some being as small as a pea, and others as large as a cocoa-nut. They may be limited to the branches of one nerve or be distributed quite irregularly. Plexiform neuroma consists of a mass of nodular, interlacing cords of varying thickness, made up of much fibrous tissue arranged in concentric lamellæ, coursing through which the nerve fibres will be found, unaltered, as a rule. The tumour is usually situated on one of the branches of the fifth nerve, in the orbit or upper eyelid, for instance, or on the cervical, brachial, or lumbar plexuses. It may grow to a very large size, although as a rule its growth is slow, but causes no painful symptoms unless by its mechanical pressure.

Tubercula dolorosa is the name given to minute, excessively painful tumours which occur on the peripheral terminations of sensory nerves. Amputation neuromata are the small, exceedingly painful growths which develop on the divided ends of the nerves in a stump after amputation. They consist of greatly twisted and contorted strands of nerve fibres mixed with some connective tissue.

Symptoms.—True neuromata may show no symptoms whatever, and only be discovered after death. Very often, however, there is acute intermittent pain darting along the branches of the nerve on which the tumour is placed. There may be tingling or numbness when the nerve is much affected, and some of the muscles supplied by the nerve may be paralysed. Reflex spasm is often produced in muscles in the neighbourhood of, or even remote from, the tumour. In the case of plexiform neuroma, there are no symptoms beyond the appearance of the tumour and sometimes local pain from its pressure. The tubercula dolorosa and the amputation neuromata give rise to exceedingly acute pain, and the latter may prevent the patient from wearing an artificial limb.

Prognosis.—The prognosis is favourable as regards life, but the pain may prove very troublesome. Neuroma of the vagus may, however, cause grave cardiac disturbances, increased by pressure on the tumour. Neuroma of the cauda equina may cause paraplegia with atrophy of the muscles.

Treatment.—Excision, where possible, is the only curative treatment. The tumours may be shelled out, or the nerve divided above and below them and the tumours removed. In the latter case the nerve may, of course, be completely destroyed. After operation the nerve endings sometimes enlarge, as in amputation neuromata, and cause bulbous growths. The tubercula dolorosa and the amputation neuromata should always be excised, but it is rarely advisable to operate on the plexiform tumour. It is impossible to excise all the nodules in multiple neuroma, but any that are painful may be removed. Nerve-stretching may give relief to the pain when other remedies have failed; and subcutaneous injections of cocaine will completely arrest it, though for a short time only.

A. BRUCE

DISEASES OF THE VERTEBRÆ.

INJURIES.

The only injuries of the spine which require to be considered here are dislocation and fracture of the vertebræ.

Simple dislocation occurs in the cervical region, most frequently at the fifth or sixth vertebra. It may be gradual or sudden, secondary to bone disease or the result of injury. The damage to the cord is always greatest in traumatic displacement of healthy bones, because of the force needed to produce such dislocation. Displacement may occur in any direction. It usually involves both vertebral articulations, but rarely may affect one only. It is caused by violent blows or falls on the head, or sudden rotation of the head while a weight is carried on it.

The symptoms are a lateral or forward or backward displacement of the head, so that the chin is in contact with the shoulder, or the occiput with the nape of the neck. The irregularity of the spine is usually readily detected. The cord is damaged in most cases, and the symptoms are those of a total transverse lesion. It may, however, be merely compressed, and the symptoms of paralysis have been known to pass away, in such a case, on the reduction of the dislocation. More commonly, the cord is bruised, and secondary myelitis occurs. In rare instances, the cord has been completely divided. Rupture of the transverse ligament, which retains the odontoid process, may permit the latter to compress the cord, and thus cause instant death. This often results from the sudden suspension of the head, as in criminal execution. Rupture of a ligament has often resulted from raising a heavy weight with the head. The treatment in dislocation is too surgical to be described here.

All organic diseases of the bones predispose to fracture, and amongst those, one has special interest in this connection. The vertebræ may share the weakening of the osseous tissues occasionally produced in tabes, and when this has taken place, a slight influence, such as a blow or a wrench, may cause fracture. The force needed is often extremely slight. Apart from disease, the accident is most common in adults, and is usually the result of a blow or a fall on the spine, or of sudden forcible flexion. It is most frequent at the fifth and sixth cervical vertebræ, and at the last dorsal or first lumbar. In the latter position the bodies are broken in most cases, but in the cervical region the arches alone are fractured in one half. Usually there is displacement which involves a narrowing of the canal and compression of the cord. But the cord may be seriously damaged when there is no such narrowing. Blood is usually extravasated outside the dura mater, often in considerable quantity, and there are small extravasations in the pia. It is more common to have local myelitis apparently the direct result of concussion. The myelitis may spread from this focus so as to affect the whole length of the cord below the injury.

There are—(1) local indications of the injury to the spine; (2) nervous symptoms, such as general shock, vomiting, etc., not distinctly due to the damage to the cord. There is great pain at the position of the fracture, rendered very intense by pressure, and often radiating along the roots. In rare cases, epileptiform convulsions have followed fracture, usually after an interval of some days. (3) The symptoms which result from damage to the cord, namely, paralysis in the parts below the injury. Its character depends on the amount of damage. Reflex action is lost at the level of the lesion, and below it is usually in excess, unless the centres are impaired by descending myelitis. The pains in the arms are severe when the fracture is opposite the cervical enlargement, and in the legs when at or below the lumbar enlargement, so as to damage the nerve roots. There is usually incontinence at first, afterwards retention of urine, and cystitis, bedsores, etc., may supervene. Ultimately, if the lumbar enlargement is not involved in the inflammatory process, there may be increased myotatic irritability in the limbs, progressing to spasm. Special symptoms result from injury in certain parts. These depend upon the parts supplied by the injured cord, and need not be described in detail. Secondary myelitis may occur at a variable period after the injury, and it and its consequences are frequent causes of death.

CARIES.

Caries of the bones of the spine is a frequent cause of paraplegia. It is often termed Pott's disease, or Pott's paraplegia, from the English surgeon who first described it as a cause of paralysis.

Etiology.—Males are said to be more liable than females. It is more common in childhood and in early adult life than any other scrofulous lesion. Injury seemed to frequently excite the mischief to the spine, although there is usually an interval, sometimes of many months, between the injury and the definite bone symptoms. Soft growths in the spine and, in rare cases, syphilitic formations have had the effect of causing breaking down in the bodies of the vertebræ. Osteitis, due to extension from adjacent inflammation, is a rare cause.

Morbid anatomy.—There is inflammation of the bodies of the vertebræ and of the intervertebral substance, often at first with enlargement, and subsequently breaking down of the substance of the bones. Products of inflammation and destruction of tissue accumulate, either inside the canal or outside the vertebral column. In the latter situation they are increased in importance by the inflammation they excite, and the well-known forms of abscess result. Within the canal the inflammatory products are of firmer consistence; the inflammation has little tendency to spread as a purulent meningitis, or even to penetrate the dura mater. The firm consistence of the inflammatory products is an important pathological feature, because this material is so often the agent by which the cord is compressed. The displacement that results from the disease varies. Occasionally there is simply a lateral displacement—one spine a little to the side of that above it. More commonly the collapse of the bodies leads to angular curvature, but the bend is often less abrupt. The nerves, as they pass through the membranes and foramina, are irritated by the inflammation, and often compressed by the thickening of the dura mater. The damage to the spinal cord is variable, and uncertain in character and occurrence. The damage is of twofold nature—compression and inflammation. The compression may be attended by chronic or acute inflammation. The mechanism by which the compression takes place varies. It is most frequently effected by the collection of inflammatory products outside the dura mater, and by the thickening of the membranes.

Less commonly, it is by the displacement of the bone, or by fragments of bone that are pushed into the canal. Often both of these factors are influential; probably an inflammatory swelling or abscess of the bone is sometimes the mechanism in compression, since the signs of pressure have disappeared when an abscess has formed outside the spine, or even when deformity has come on through collapse of the bodies.

Symptoms.—The symptoms are of three kinds. First, those due to the bone disease; secondly, those due to damage to the nerve roots; and thirdly, those due to the change in the cord itself. The bone symptoms, although not strictly within the sphere of nervous disease, are of much importance, as on them the diagnosis of the cause of the nervous symptoms often depends. This gives the subject its high medical importance, and brings consideration of the bone disease and its symptoms within the province of practical medicine.

Due to bone disease.—The first is pain in the spine, chiefly at the affected spot, increased by movement, and especially by pressure. The increase of pain by movement is greatest when the disease is in the more mobile parts of the vertebral column, especially when it is in the cervical region. Deformity of the spine is a later symptom than the tenderness, and usually comes on gradually. It is often absent when the disease is in the cervical region; but in such cases there is usually another symptom, namely, thickening of the tissues about the spine, which is rare in other parts. In any region an abscess may form in the neighbourhood of the disease. Those that form in front of the diseased vertebræ are especially puzzling if the existence of spinal caries is not known. Increased pain and tenderness in the vertebral column often precede the indications of damage to its contents.

Due to interference with the nerve roots.—These are very variable, and may be severe or absent. There may be hyperæsthesia along the course of the nerves that emerge at the affected region. There is also muscular weakness and wasting. The motor symptoms are most marked when the disease is in the cervical region, and the nerve roots for the arms are damaged. Herpes zoster has been occasionally met with along the course of the irritated nerves. Spasmodic contraction of the muscles supplied by the roots is rare.

Due to cord disease.—These are the results of interference with the conducting function of the cord, which gives rise to paralysis below the lesion. Usually there is no exciting cause for the onset of cord symptoms, and when they have commenced they may develop quickly or slowly. Very rarely the onset is spontaneous, probably from sudden displacement. Both legs usually suffer together, and occasionally one is paralysed before the other. In the most frequent cases the dorsal cord is damaged, and the condition of the legs is the familiar one of spastic paraplegia. If the disease is situated in the cervical region, there may be muscular atrophy in the arms, as well as spastic paralysis in the legs. Sensory impairment due to disease of the cord is less common than motor palsy; the onset of the paralysis may be preceded by subjective sensations in the legs. Reflex action is excessive, unless the disease involve the lumbar enlargement. The sphincters are often affected, sometimes early, but they may escape, even when there is motor palsy of the legs.

Amongst the common complications are bedsores, cystitis, abscess in connection with bone disease, tuberculous disease elsewhere, and general tuberculosis. In very severe cases, secondary mischief may spread from the local seat of disease in the spinal cord, either as a descending myelitis, or as an ascending myelitis. Ataxy may come on as power returns, and myelitis may occur in disseminated foci in various parts of the cord, or even in the medulla.

Course.—The cord mischief is influenced by the state of the bone disease. Pressure on the cord may be relieved although the bone disease continues, so that occasionally angular curvature may develop as paraplegia passes away. In some cases paralysis, both motor and sensory, persists. More often the sensory loss passes away, while motor impairment remains in the form of spastic paraplegia. But in many cases the paralysis gradually passes away, and in children recovery occurs far more readily than in adults. Even in adults, however, recovery may occur, although the paralysis may have lasted for more than a year, with all the signs of severe spastic paraplegia. Usually rest on the back or mechanical supports are necessary to secure recovery, but it occasionally occurs without those measures. Relapses sometimes occur in cases that improve, although not so frequently as might be expected. In several cases, the subjects of caries in early life have at some period in adult life presented the symptoms of primary lateral sclerosis. If there is any connection between the two, it is probable that the cord has suffered compression so slowly that its functions have not been interfered with, but nevertheless the vitality of the pyramidal fibres has been rendered less enduring. It is important to remember that the curvature is often lateral, and also that slight lateral deviation or slight prominence is not unusual in normal spines.

Diagnosis.—When the signs of caries coincide with the presence of paralysis, the diagnosis is comparatively easy. The cases which present most difficulty are those in which the paralysis is present without obvious deformity in the back, or other symptoms indicating bone disease. Regard in such cases must be had to the slight bone symptoms,—especially the deep tenderness,—and perhaps slight irregularity, which are so often present. An observed increase in this irregularity is, of course, of extreme significance, and so are root-pains at the point where the deformity and the tenderness are distinct. When damage to the cervical arches causes muscular wasting in the arms, the case may resemble one of progressive muscular atrophy, but it will probably differ in the affection of sensation, and in the presence of pains. When there are merely tenderness of the spine, with slight weakness of the leg, the question may arise whether there is organic disease, or merely the condition termed spinal irritation. In these cases, however, the tenderness is usually much more widely distributed, it is superficial as well as deep, and there are no root-pains or spots of anæsthesia. There is more danger that caries of the spine in a young woman may be regarded as hysterical paraplegia than that the opposite error should be made. And it should never be forgotten that patients, especially young women, with unequivocal signs of caries, may have, in addition, all the symptoms which are usually ascribed to what is called hysteria.

Prognosis.—Our ignorance of the precise character of the morbid process which is damaging the cord renders the prognosis in every case a matter of much uncertainty; but there is no disease in the cord in which symptoms so severe so frequently pass away. The cases are few in which hope is unjustified. In childhood the prospects of recovery are certainly better than in adult life; in old age the prognosis is bad, and the disease nearly always has a fatal termination. Damage to the cord between the enlargements is less serious than when one or other of those is affected. The danger to life is dependent in considerable degree on the evidence that may be present that tuberculous disease is active elsewhere, and the prognosis is further modified by the recent addition of caries to those diseases which may be attacked by surgical means.

Treatment.—For directions as to the treatment of the bone disease, the reader is referred to treatises on Surgery. If the bone disease heals, the spinal cord, in most cases, will recover. Two most important means of treatment are persisent recumbency, and tonics, especially cod-liver oil and iron. In the cases that have done best, these and these alone have been used. The best posture is upon the back, and if no improvement occurs after some months' rest, suspension may be tried. In caries of the cervical spine, fixation of the head is necessary, even during the period of rest. Counter-irritation opposite the seat of caries sometimes has appeared to do good. The actual cautery in mild form has been found most frequently useful. Sulphide of calcium, which is said to influence scrofulous processes, deserves further trial. Extreme care in general management, to avoid bedsores, bladder troubles, bronchitis, and other complications, are of great importance. When power has returned, but certain contractures have taken place, the ability to stand and walk may be quickly restored by tenotomy. Electricity is useful only to maintain the nutrition of muscles that are wasting, and the same may be said of gentle upward rubbing. The efficacy of operation in those cases is probably not so great as it was supposed to be a few years ago. Certain cases undoubtedly do well; in others, however, little or no improvement has been effected, and in another class recovery has followed operative treatment, but recurrence has taken place, and the case has ended fatally. Operation is certainly contra-indicated if there are signs of rapidly-advancing general tuberculosis. It is also contra-indicated in cases in which the disease has lasted a long time, and in which there are secondary changes in the kidneys, liver, or other organs. The prospect of recovery, however, is excellent if the bone disease has become quiescent, or the caries is actually healed. In such cases, the removal of pressure on the cord may be followed by a rapid and complete recovery, the pressure presumably being due to the mass of connective tissue which in these cases forms a distinct tumour on the outer aspect of the dura mater. The chief obstacle to success is the occurrence, in certain cases, of severe myelitis at the seat of pressure—a myelitis quite out of proportion to the degree of pressure which has been present. This, of course, no removal of pressure can relieve. To justify surgical interference, the existence of caries must, of course, be beyond doubt. The operation involves some danger to life, and this fact ought unquestionably to receive due consideration.

TUMOURS.

The bones of the spine are sometimes the seat of primary or secondary growths, and are sometimes invaded by tumours springing from contiguous structures. Cancer and sarcoma are most common; myxoma has also been met with. A primary growth usually begins in the bodies of the vertebræ, and spreads from one to the other. From the bodies the growth may extend into the lateral processes, enlarging them, and narrowing the intra-vertebral foramina. The nerve roots may suffer by pressure, simple inflammation, or cancerous infiltration. The cord may suffer compression, and is often inflamed, sometimes acutely. Such inflammation, once excited, may spread in the cord, upwards and downwards.

Etiology.—The general causes are the same as those of similar growths elsewhere, and injury has been sometimes supposed to be the exciting cause.

Symptoms.—There may be direct symptoms—pain in the spine, local tenderness, and interference with the movements of the vertebral column. A palpable tumour is never an early symptom; but ultimately a deep-seated hard swelling may be felt on one side of the spine. Angular curvature sometimes occurs, similar to that of caries in its external characters. The affection of the nerves is almost invariable, and gives rise to the most distressing symptoms, with radiating pain in the course of them. At first slight, these gradually increase to extreme intensity, and even slight movement of the trunk induces the paroxysms. It is rarely that those pains are absent, but they occasionally occur late. But, on the other hand, they may precede other symptoms for months. Corresponding damage to the motor roots may cause contracture, paralysis, and wasting. The damage to the cord causes symptoms similar to those in caries, and described under "Compression." The chief difference from caries is in the sudden onset of paraplegic symptoms, due to the onset of acute inflammation. All power in the legs is often lost in twelve or twenty-four hours, even when no curvature has taken place. On the other hand, the onset of the palsy may be very slow, and it is known to occupy several years in reaching a considerable degree. The characters of the resulting paralysis are, as a rule, similar to those in caries, but are more frequently modified by the spread of inflammation; so that, for example, the central and reflex functions of the lumbar enlargement may be lost at the onset. In many cases, however, there is no descending inflammation.

The course of the disease is naturally progressive. Sometimes, however, some improvement succeeds a rapid development of palsy, due to the partial recovery from the state of acute inflammation. The duration of the disease varies according to the nature of the growth. Death may be due to bedsores, cystitis, and kidney disease, to growths elsewhere, or to simple exhaustion.

Diagnosis and prognosis.—The recognition of the disease is only a matter of certainty when signs of tumour are present, or when they occur in a patient from whom malignant disease has been removed. It is important to remember that acute transverse myelitis may be an early effect of the disease; and the presence of this condition, with marked local tenderness, is very suggestive. It should, however, be remembered that similar root symptoms may be produced by an aneurysm, or a growth in front of the vertebral column. The symptoms of a growth in the lower cervical region may be closely simulated by the severe root pains, the result of radicular neuritis. The distinction rests on the absence of the signs of a growth, but it is usually necessary to wait and watch the course of the symptoms. The chief difficulty in diagnosis is the distinction from caries. One distinction which is suggestive, but not absolute, is the intensity of the pain in tumour, and especially its increase on movement. Among other diseases with which these growths may be confounded is the dorsal form of tabes; but the wide extent of the root symptoms, the slight effect of movement, and the loss of the knee-jerk, are usually sufficient for distinction. The prognosis scarcely requires formulating, the only point requiring determination being the probable duration of life; and judgment on this matter must be formed from the severity of the symptoms and the constitutional condition.

Treatment.—Possibly, in a few cases, the growth may be so placed as to be removable, and an exploratory operation would be justifiable in any promising case. But treatment usually has to restrict itself to an attempt at the relief of pain, and to the avoidance of bedsores and other results of the disease. Morphine is alone powerful for the relief of pain; but unfortunately the dose has to be quickly increased. Cocaine, however, affords some relief, and may, with other anodynes, at least prolong the effect of morphine.

W. R. GOWERS.
JAMES TAYLOR.

DISEASES OF THE SPINAL MEMBRANES.

THE morbid processes that involve the membranes are chiefly hæmorrhages, growths, and inflammation. Tumours of the membranes may be conveniently considered with those of the spinal cord. Inflammation and hæmorrhage are described here.

MENINGITIS.

Inflammation of the membranes may be acute or chronic, may chiefly affect the dura mater (pachymeningitis) or the pia mater (leptomeningitis). The arachnoid usually suffers with the pia, but is said, in one form of inflammation, to be affected almost alone, and this form has been termed arachnitis. Acute inflammation beginning in one membrane usually spreads to the others. Chronic inflammation may remain limited to one. Spinal meningitis may be divided into two forms—(1) that which begins outside the membrane, often arising by extension from some adjacent focus of inflammation—external meningitis; (2) that which begins within the sheath of the dura mater, and is often primary—internal meningitis.

EXTERNAL MENINGITIS.

External meningitis is thus an inflammation of the dura mater. It also involves the connective tissue outside the membrane, and these may suffer before the membrane itself. The morbid process usually remains limited to the outer surface of the dura, passing through it only in rare cases.

Etiology.—External meningitis is usually the result of extension from some contiguous disease, especially caries of the spine. A syphilitic inflammation may, in rare cases, be associated with syphilitic disease of the vertebræ. Such external meningitis is usually limited in extent and chronic in course. Deep sacral bedsores, however, sometimes cause an acute inflammation, and ascending neuritis has been supposed, in some cases, to set up local inflammation. It is certain that acute external general meningitis occurs as a primary disease, and may run an intensely rapid course, with profuse suppuration between the membrane and the bone. The subjects have generally been young, ill-nourished adults, and there is often a history of exposure to cold. In some cases the pus has worked its way backwards, and has spread amongst the muscles of the back, forming collections at one or more spots, either in front or behind.

Pathology.—The inflammation is sometimes simple, much more frequently purulent. In the former case, the dura mater may be merely reddened and opaque; in the latter the surface is covered with a layer of pus. In the more common, a layer of purulent inflammatory products covers the outer surface of the dura mater. Often the inflammatory products undergo development into fibroid tissue, so as to give rise to the actual appearance of a tumour. Sometimes there is a regular nodular thickening of the outer layer of the membrane; but in most cases the inner surface is normal, even when the cord itself is inflamed. In all forms of external inflammation, the fat outside the dura mater is quickly absorbed, and the membrane may become adherent to the bone. When secondary to caries, the inflammation is usually limited to the neighbourhood of the bone disease. When it is acute and general, pus may spread through the whole length of the canal, and, as has been stated, may often be found among the muscles of the back. In such cases the membrane is swollen, sodden, and in appearance resembles a piece of wash-leather. The inner surface is usually normal.

Symptoms.—These vary considerably. They have been already described in association with caries. The chief symptoms are pain in the back, often referred to the loins, increased by movement, and these pains are usually associated with spasm. The hyperæsthesia may be followed by anæsthesia, and sometimes there is paralysis of the muscles from involvement of the motor nerve roots. The sphincters are usually paralysed, and in acute cases there is much constitutional disturbance, rigors, profuse sweating, and high fever. In local secondary forms the symptoms differ with the acuteness of the inflammation. Although pain, as a rule, is considerable, other signs of irritation are inconspicuous.

Diagnosis and prognosis.—The symptoms of irritation of the nerve roots constitute the most important indications, combined with the signs of pressure on the cord. The acute form causes symptoms that have much in common with those of internal meningitis, and it is doubtful whether a diagnosis can be made by the character of the nerve symptoms alone. In a case of apparently primary meningitis, it is important to watch carefully for any signs of deep œdema among the muscles of the back. This will indicate commencing purulent inflammation extending from within, and might be accompanied by signs of inflammation in the pleura, in the peritoneum, or behind the pharynx, from a similar extension in the other direction. The acute affection is exceedingly grave. Recorded cases have ended in death, but it is possible that some cases have recovered, and that the fatality of the disease may not be so great as would appear from the published facts. Chronic external meningitis resulting from caries is only serious by reason of the compression it exerts on the cord.

Treatment.—The local form alone affords opportunity for effective treatment. Any accessible collection of pus should be opened, and if acute local symptoms suggest the passage of pus into the cerebral canal, the propriety of operation should be considered. Rest, counter-irritation of the spine, especially by the actual cautery, sedatives to relieve the pain, and tonics, are the most important measures in chronic cases. So far as the acute form is open to treatment, the measures suitable are those for the internal variety. In a subacute case, a free exit should be allowed for pus, but an opportunity for this seldom presents itself.

ACUTE INTERNAL MENINGITIS.

This, for the reason already mentioned, has been termed leptomeningitis, but the acute form only remains limited to those membranes when of slight degree. In some cases, the inner surface of the dura is also inflamed. Frequently the inflammation extends to the cord, such cases being very often termed meningo-myelitis. In other cases, curiously enough, no extension to the cord seems to take place. The inflammation may be either simple or purulent, or may be secondary to tubercle. It also occurs in conjunction with cerebral meningitis in the epidemic form. Acute simple spinal meningitis is rare. The immediate causes are, in part, such as have a local action, and general morbid influences.

The local causes may be—(1) injuries to the spine; (2) exposure of the back to cold, or to the sun, has seemed to be effective in certain cases; (3) adjacent inflammation has sometimes caused internal meningitis, but in such cases external meningitis has been a necessary preliminary.

In a few cases, internal meningitis is due to the extension of inflammation from the cerebral membranes. Such extension is rarely traced, except (1) in cases in which slight meningitis is found in the upper cervical region, as far down as gravitation favours the descent of solid particles in the cerebro-spinal fluid; and (2) in cases in which some cause has been effective in producing inflammation of the cerebral membranes, and, later, the affection of the spinal membranes, such as the local presence of tubercles, or a morbid blood state.

The most severe forms of internal meningitis are due to a morbid blood state, either septicæmia or a virus allied to that which causes the cerebro-spinal form. In cases of acute inflammation, organisms have been found in the spinal fluids. General exposure to cold probably causes an effect only through the agency of toxæmia. Such exposure has apparently been rendered effective, in some instances, by coincident menstruation. The disease also may occur in the course of, or subsequent to, certain acute specific diseases.

Morbid anatomy.—It is usually wide in extent, as the inflammation spreads readily in the loose tissue in the arachnoid. In the earliest stage the change only consists of congestion in the pia, perhaps, also, the inner surface of the dura and substance of the cord. In a more advanced stage, the pia arachnoid is opaque and thickened, and an exudation of inflammatory products may be present. This exudation may be semi-purulent, and the inner surface of the dura mater may present similar changes. The vessels are dilated and their sheaths distended with cells; the spinal fluid is increased in quantity, turbid or even purulent in aspect. The nerve roots also may be covered with exudation, and the spinal cord itself is often invaded. The change is always most marked in the periphery. If recovery take place, the membranes may remain opaque and adherent, and there may be a permanent excess of arachnoid fluid. The changes in the cord may lead to sclerosis. In tuberculous inflammation, the amount of exudation is usually small; it is often grey and gelatinous, and in it are scattered the tuberculous granulations. In many cases the signs of spinal meningitis are associated with those of inflammation of the membranes of the brain, and a continuity of the processes may be obvious or indistinct. In the violent purulent cerebro-spinal meningitis, which is occasionally produced by a septic influence, pus around the cord is often continuous with that which bathes the base of the brain.

Symptoms.—Malaise, with slight pain in the back, may precede the acute onset of symptoms. This is usually marked by a rigor, hyperpyrexia, and a severe pain in the back. Pain may also radiate along the distribution of the nerve, and the eccentric pain is paroxysmal and intense. Pain in the back is increased by movement, also by pressure, and it is no doubt due to irritation of the inflamed meninges. Muscular spasm is usually coincident with pain. There is rigidity in the muscles, most marked in the neighbourhood of the inflamed part. It is often first observed in the neck. When slight, it may merely cause retraction of the head, or stiffness of the back; or if severe, may even cause opisthotonos. It usually involves also other trunk muscles, especially those of the abdomen, and the limbs may even become rigid. There is usually great hyperæsthesia of the skin, and increased sensitiveness in the muscles. Reflex action is increased, and constipation and retention of urine are common. Dyspnœa may result from spasm in the thoracic muscles. The pulse may be frequent, or retarded, and headache, delirium, or coma may occur when the inflammation has extended within the skull. The temperature is raised—sometimes, however, to only a slight degree. Cheyne-Stokes breathing may be present from implication of the medulla. The function of the vasomotor nerves is deranged.

In time, the symptoms of irritation give rise to those of paralysis, the limbs become lax, sensibility becomes lessened or lost, and reflex action disappears. The heart is apt to become feeble, and death may occur from asthenia, or from paralysis of the respiratory muscles. Towards the end, there is sometimes a considerable rise of temperature. Death may occur from the effects of bedsores, or from secondary kidney disease. But, on the other hand, the signs of irritation may lessen and disappear, while those of structural damage remain. The persistent symptoms vary much in extent and degree, and consist in paralysis—motor and sensory—with added muscular atrophy and contracture. Ultimately, the symptoms of damage to the cord may either slowly increase, or, on the other hand, those of meningitis may pass away entirely. The symptoms vary in their distribution according to the position of the disease—in the lumbar, dorsal, or cervical region. When the latter region is affected, the symptoms are manifest in the upper extremities, the dyspnœa may be great, the action of the heart may be deranged, and the pupils may be affected. Extension to the brain is marked by vomiting, general headache, delirium, and affection of the cranial nerves.

The symptoms vary also according to the nature of the inflammation. In purulent meningitis, the symptoms are sometimes very slight, and the duration of the acute symptoms varies from a day or two in severe cases, to two or three weeks in cases of less severity. The symptoms of tuberculous spinal meningitis resemble those which have been described, but are usually less intense.

Diagnosis.—The diagnosis rests on the symptoms—the pain in the back, the retraction and rigidity of the neck and spine, hyperæsthesia, and spasm in the limbs. In myelitis, pain in the back is absent or trifling. Meningeal hæmorrhage most nearly resembles meningitis in its manifestations; the distinction depends chiefly upon the mode of onset. Hæmorrhage into the cord can scarcely be confused with inflammation of the membranes. Tetanus sometimes presents a close resemblance, but there is no fever at the onset, and in this disease trismus is an early and obtrusive symptom, and the paroxysms of muscular spasm are excited by peripheral impressions. Rheumatism of the muscles may cause pain of the back on movement, but there is no spontaneous pain, such as attends meningitis, nor is there distinct spasm. The diagnosis of the form of meningitis depends on the recognition of the cause of inflammation. When spontaneous and acute, the case is probably the primary form. If it occurs under conditions which give rise to surgical septicæmia or after childbirth, purulent meningitis is almost certain. The diagnosis of the tuberculous variety depends on the pressure of a combination of the symptoms of cerebral meningitis, and on the gradual and insidious onset.

Prognosis.—This is grave in all cases. It is worse when the symptoms are severe and acute, and the temperature is high. It is also worse when it is due to serious lesions of the spine; and is worse in the so-called spontaneous cases than in those which result from traumatism. Recovery is more probable in middle than in early or advanced age.

Treatment.—Perfect rest and quiet are of the utmost importance. Although it is undesirable that the spine should be the lowest part of the body, yet in acute meningitis it is scarcely possible for the patient to lie in any other posture. Dry, or in robust patients wet, cupping or leeching along the spine may be employed at the onset. Counter-irritation by blisters or by mustard is more useful when the disease is subsiding. Hot-air, or vapour baths in cases which are due to cold, should be employed at the onset of treatment. Mercury is the only internal remedy which has been regarded as capable of influencing the inflammatory process. It should be given until there is a slight affection of the gums. The oleate of mercury may be rubbed in along the spine. Iodide of potassium seems to have little influence. It is usually necessary to give sedatives for the relief of the pain, and of these, morphine by the skin or by the mouth is the most effective. Sometimes inhalations of chloroform are necessary, and may be even more effective than morphine. In slight cases, relief may be effective by belladonna, or by atropine hypodermically. Chloral, or chloral and bromide, or any of the milder sedatives, may be given if there is insomnia. When the disease has passed into a chronic stage, iodides have been thought useful. Counter-irritation may then be employed freely with advantage. Tonics, such as iron and quinine, and even strychnine, are beneficial. Warm baths, such as those at Bath and Aix-les-Bains, seem sometimes of service, especially when employed as hot douches. The local consequences, such as muscular atrophy, contractures, and the like, need special local treatment, by rubbing, electricity, and similar measures.

CHRONIC INTERNAL MENINGITIS.

This occurs in two forms—pachymeningitis and leptomeningitis. They have many characters in common; they own the same causes, and need the same treatment; hence it is most convenient to describe them together. Chronic meningitis is rare as a general and primary malady, and is seldom fatal. There is little doubt that the condition which twenty years ago was looked upon as chronic meningitis was really often primary lateral sclerosis.

Etiology.—It is most frequent in adult age, affects men more frequently than women, and has been thought to occur more readily in patients with neurotic heredity. Debilitating influences predispose to it; and among exciting causes are severe and repeated exposure to cold, traumatic lesions, concussion, etc. It may also result by spread from without inwards, and by extension from the substance of the cord itself. Local chronic meningitis may result from any chronic disease of the membranes, the bones, or the spinal cord. It occurs in all cases of compression, and in all forms of myelitis which involve the superficial layers of the cord. Certain general morbid states are frequent causes. Of these, that due to alcohol is perhaps most important. The second common cause is syphilis, which may produce either pachymeningitis or local leptomeningitis, irregular in position and effect. Tubercle also has given rise to a chronic inflammation chiefly of the inner surface of the dura. Hæmorrhagic pachymeningitis occurs especially in the insane, but has been met with as a result of alcoholism and of injury.

Morbid anatomy.—In slight cases there is merely thickening and opacity of the membrane, with perhaps distension of vessels. The spinal fluid is increased in quantity, and is turbid. In more severe conditions, the dura and pia may be connected together by a layer of inflammatory tissue, and the microscope shows the ordinary elements which result from inflammation. Frequently also the pia is transformed into a thick, irregular layer of homogeneous tissue, without any distinct cell elements. The walls of its vessels may be greatly thickened by similar material. The nerve roots passing through the diseased membranes very seldom escape. The fibres suffer especially when the inflammation involves the dura; and when the pia mater is thickened, scattered tracts of connective tissue may be seen in the substance of the nerve roots between the fibres.

The spinal cord may be little affected, but is frequently damaged by the extension to it of the inflammation of the pia. Ultimately this results in the formation of indurated tissue, constituting an irregular zone of peripheral sclerosis, and from the thickened pia tracts may extend into the cord, sending out branching trabeculæ. The peripheral sclerosis associated with chronic meningitis frequently damages tracts that undergo secondary ascending degeneration. The result is to produce a mixed lesion, the precise nature of which it is not always easy to distinguish. This difficulty is increased by the fact that a similar thickening of the pia occurs over the areas of the cord that are the seat of other forms of sclerosis, both those that are systemic, such as tabes, and those that are random, as the insular form.

Inflammation beginning in the pia mater and arachnoid is often extensive in range, and may be considerable in degree, or slight. That which commences on the inner surface of the dura mater is frequently limited in extent, but is accompanied by the formation of a large amount of new tissue, hence it has been termed hypertrophic internal pachymeningitis. This is a very important variety, and gives rise to peculiar and grave symptoms. It is most common in the cervical region, and consists in a great thickening of the inner part of the membrane, sometimes as much as a quarter of an inch. Several layers of new tissue may often be distinguished. The pia may be normal, the cord is compressed and commonly softened at the spot, and presents signs of inflammation. The nerve roots are also damaged. In rare cases the membranes about the cauda equina may be alone affected. The dura and pia are adherent, and the nerves united in a fibrous mass. The white fibroid plates found so often in the arachnoid after death are probably not connected with the preceding inflammation. It is said, however, that when they are numerous and extensive, they may give rise to symptoms closely resembling those of chronic meningitis.

The rare condition termed internal hæmorrhagic pachymeningitis, or hæmatoma of the spinal dura mater, is similar to and commonly associated with that which affects the cranial membrane. In this a reddish brown exudation covers its surface, composed of fibrin and extravasated blood, the latter of which may be encysted in small cavities, and in various stages of transformation. The local form of syphilitic meningitis presents irregular thickening of the pia and arachnoid. The tissue has a peculiar blue aspect, and presents a tendency to caseous degeneration and fibroid change. At some spots the growth may attain such a development as to constitute a distinct gumma. A similar process may be sometimes traced into the substance of the cord along the tracts of the neuroglial tissue and the periarterial spaces. The whole thickness of the walls of the arterioles may be the seat of inflammatory thickening, leading to constriction, and even obliteration of the arteries. There seems also reason to believe that a similar condition of vessels may occur apart from syphilis.

In the very rare chronic tuberculous meningitis, the dura mater is covered with a layer or layers of tubercle on its inner surface. The arachnoid may also contain degenerating tubercles, and the two membranes may be united by the new formation.

Symptoms.—The symptoms are the result of irritation of membranes, of damage to the nerve roots, and of the cord. Spasm, which dominates the aspect of acute meningitis, is almost entirely absent in the chronic form. Common symptoms are pain in the back, increased by movement and by pressure on the vertebral spines. Radiating or eccentric pains, due to the irritation of the nerve roots, referred to the back of the head, neck, arms, thorax, abdomen, loins, and legs. Hyperæsthesia may also exist, and cutaneous eruptions have been observed in association with the pains. Muscular twitching, tremor, or even spasm, may result from irritation of the motor root fibres. After a time, certain paralytic symptoms manifest themselves, depending on further damage to the nerve roots. Sensation becomes lessened, and the muscles waste. The wasting is often irregular. Reflex action in these parts is lost, and as the disease progresses, the cord itself suffers by compression, or by extension to it of the inflammation. The parts below the seat of the disease are then paralysed; reflex action in them may be increased, their muscular nutrition may remain good, but defect in co-ordination is not uncommon. Power over the sphincters may be lost, and trophic changes may occur, especially if the disease is over the lumbar region. When the inflammation chiefly affects the pia and arachnoid, the symptoms of meningeal irritation are conspicuous. The local paralytic symptoms may be absent in those cases. On the other hand, weakness and pains in the legs may be early symptoms. When the inflammation begins in the dura, the symptoms of irritation, although occasionally present, are much less prominent symptoms. The radiating pains are severe, and at a later stage the muscular weakness chiefly attracts attention. This is especially the case when the lesion is in the cervical region—the cervical hypertrophic pachymeningitis described by Charcot and Joffroy. The wasting may involve many muscles; those supplied by the radial nerve are usually less affected than the others. With this muscular weakness there is paraplegic weakness, and in rare cases the disease affects the membranes over the lumbar enlargement, and then the legs are the seat of pains and paralysis, as already described. The sphincters are early paralysed. The most frequent and special characteristic of chronic syphilitic meningitis is the focal character of the symptoms, and their tendency to be associated with those of partial and local damage to the spinal cord itself. In hæmorrhagic pachymeningitis the symptoms are those of a slight chronic inflammation, without any distinct features that can be ascribed to the hæmorrhagic condition.

Diagnosis.—The spinal pain and stiffness may be mistaken for spinal irritation, or for simple spinal pain. But in the former the whole spine is tender, and pain in the back is not related in such degree to posture and exertion. Peripheral pains are prominent symptoms in some cases of muscular rheumatism, but in this there are not the pain and tenderness of the spinal column. Radicular neuritis is probably associated in many cases with some affection of the adjacent membranes. The muscular wasting may be mistaken for atrophy due to degeneration or subacute inflammation of the anterior horns. And the cervical hypertrophic pachymeningitis bears a special resemblance in this respect to progressive muscular atrophy, except for the pains and anæsthesia. Chronic disseminated myelitis may cause muscular wasting, but does not cause pain in the back or rigidity of the spine. It must, however, be remembered that the two conditions of meningitis and myelitis are often combined. When the meningitis is lumbar, the pains in the legs may resemble those that occur in locomotor ataxy; and the resemblance of the two diseases may be further increased by the loss of the knee-jerk. The anæsthesia and loss of the trunk reflexes are more general and uniform in tabes than in meningitis. Caries of the spinal bones produces many of the symptoms by the aid of chronic meningitis; hence the diagnosis depends on the recognition of the presence of bone disease. Multiple neuritis presents nerve symptoms of similar character to those of chronic meningitis, but the disease is symmetrical in its distribution. There is the atrophic palsy and the presence of tenderness along the nerve trunks.

Prognosis.—Severe degrees of the affection are attended with danger to life, and even the slighter forms may entail serious and lasting consequences. In many cases the symptoms ultimately pass away, and the prognosis is most favourable to those which result from injury and from syphilis. The prognosis is better in other cases in proportion to the general health of the patient, to the moderate degree of the mischief, and to the extent to which its causes are under control.

Treatment.—Rest is essential, and in severe cases should be absolute. Posture is very important; the prone couch, impracticable in acute meningitis, may often be employed in chronic cases with advantage. Warm baths are useful, especially in subacute cases. Next in importance to rest and posture is counter-irritation, repeated sinapisms, or stimulating liniments, and blisters may be used; but by far the most efficacious means of counter-irritation in this disease is the actual cautery. Sedatives, such as opium, morphine, chloral, Indian hemp, or cocaine, may be employed to relieve the pain; but it must be remembered that these will probably be needed continuously for many months, and the dose to begin with should be small, and the drug frequently changed. Iodide of potassium and mercury are the agents that have the most influence over the morbid process itself. Both are effective in syphilitic cases, but in other forms mercury has far more influence than iodide. It may be rubbed in over the spine, either the blue ointment or the oleate; and these may, if necessary, be rendered counter-irritant by the addition of cantharides ointment. In many cases, tonics such as iron and quinine are needed. Muscular wasting requires treatment by galvanism, rubbing, and by passive movement, to prevent contractures. If hæmorrhagic pachymeningitis is suspected, ergotin may be used, in addition to the other measures described. Treatment, however, appears to exert little influence upon it. Other measures to be used are those described in the treatment of the acute condition.

HÆMORRHAGE.

This may be extrameningeal or intrameningeal, and the latter may be either subdural or subarachnoid. Extrameningeal hæmorrhage is more common than hæmorrhage inside the dura mater.

Etiology.—It may occur at all ages. In newly-born children it is the result of rupture of vessels during birth. Spontaneous hæmorrhage is unknown in early childhood, but occurs at all other ages. It is occasionally found after death from severe convulsions. Muscular spasm resulting from meningeal hæmorrhage may sometimes have been regarded as primary—the cause of the hæmorrhage when it was really the effect. This seems certain in many cases of tetanoid spasm in newly-born children. Such cases should be separated from those to which the term tetanus neonatorum is applied. Hæmorrhage also occurs in some cases in which there is a hæmorrhagic tendency, and also in the hæmorrhagic forms of some acute diseases. A rare cause is the bursting of an aortic aneurysm into the spinal canal, or the rupture of an aneurysm of a vertebral artery.

Morbid anatomy.—Extradural hæmorrhage is usually not of large extent, and the blood collects chiefly on the posterior aspect. It is most common in the cervical region. The blood is coagulated wholly or partially, the dura mater is bloodstained, and the spinal cord may be compressed. The extravasation may be large or small. In subarachnoid hæmorrhage, the blood usually comes from the vessels of the pia mater. Blood effused into the spinal arachnoid may ascend so high as even to penetrate the cerebral ventricles. In meningeal hæmorrhage, cerebral as well as spinal, the spinal fluid is often bloodstained; the cord may be discoloured and compressed, and is especially damaged in the case of subarachnoid hæmorrhage. In cases which have lived a few days, there are usually signs of commencing meningeal inflammation.

Symptoms.—Sometimes no symptoms may have been observed of a hæmorrhage which is found after death. In cases in which the hæmorrhage causes symptoms, these are mostly those of meningeal irritation. The first indication of the lesion is usually sudden violent pain in the back, accompanied by pain along the course of the nerves, with hyperæsthesia. Muscular spasm usually accompanies the pain, and involves the vertebral muscles, and the muscles supplied by the nerves in which the pain is felt, as well as the muscles supplied by the cord below the seat of hæmorrhage. Paralytic symptoms quickly follow. Weakness and reduced sensitiveness in the limbs below the lesion are also found. But the symptoms necessarily differ according to the seat of the disease. The cerebral functions are unaffected, as a rule; but occasionally consciousness is lost for a short time, apparently as a result of shock. Delirium or coma may come on; but in most cases in which cerebral symptoms co-exist with those of spinal hæmorrhage, the former have been due to a simultaneous intracranial condition. In rare cases the symptoms of spinal hæmorrhage have come on insidiously, but in most cases the symptoms reach their height in two or three hours. Death may occur when the effects are fully developed; or slight subsidence may follow, and later symptoms of a secondary meningitis lead to a fatal issue. If the inflammation is moderate, however, the symptoms may subside after lasting a week or ten days. The period of the disease at which death is most common is a few hours after the onset.

Diagnosis and prognosis.—This depends upon a combination of sudden local pain in the back, with the evidence of irritation of the membranes, nerve roots, and cord. In hæmorrhage into the substance of the cord, vertebral pain is much more frequently absent than in meningeal hæmorrhage. Meningitis is distinguished by the more gradual onset of the symptoms, and by the presence of fever from the first. Diagnosis from tetanus depends on the absence of trismus, and the presence of severe spinal pain, and on the sudden onset. In all severe cases, death in a few hours is to be feared. The prognosis only becomes less grave when the symptoms have distinctly reached their height. Hæmorrhage in the cervical region is especially serious, because of the direct involvment of the mechanism of breathing.

Treatment.—The first point is to secure absolute rest; even passive movement should be avoided. The posture should be on the face or side, not on the back. The wisest treatment, probably, at the outset is scarification beside the spine, opposite the seat of pain. This may be followed by the application of ice, and by the administration of ergotin by the skin or mouth. Afterwards the bowels should be freely opened, and sedatives are usually necessary to relieve the pain. In extrameningeal hæmorrhage, it seems justifiable to remove the blood which is pressing the cord by opening the canal; and even when the blood is within the dura mater, operation seems to deserve consideration when life is threatened.

W. R. GOWERS.
JAMES TAYLOR.

DISEASES OF THE SPINAL CORD.

ACUTE ASCENDING PARALYSIS.

Paralysis which commences in the legs and ascends to the muscles of the trunk, the arms, muscles of the neck, the diaphragm, and the pharynx, is a feature of several diseases, but it occurs also in cases in which the spinal cord after death appears healthy. To such cases the name acute ascending paralysis has been provisionally applied, and from the fact that it was first described by Landry, it has also been called Landry's paralysis. It is a most formidable malady, usually proving fatal in a few days. Its nature is mysterious, and some observers have been led to the opinion that it is an affection of the nerves; but it is not probable that this opinion is correct.

Etiology.—It occurs chiefly between 20 and 40 years of age, and affects males more than females. Severe exposure to cold has been the apparent cause in many cases. In other cases the onset has occurred during convalescence from some general disease, and has followed febrile diseases of obscure nature, usually at an interval of a week or a few weeks. It also sometimes succeeds some traumatic inflammation, or an illness like an attack of pelvic cellulitis. It has been repeatedly observed in the subjects of syphilis, and has apparently been arrested in some cases by the treatment for this disease.

Pathology.—As a rule, the most careful examination has failed to discover any morbid appearance of the spinal cord, nerves, or muscles. In a few cases, apparently similar to the others, slight scattered indications of inflammation have been found in the grey matter. Changes have also been found more recently, both parenchymatous and interstitial, in the peripheral nerves. On the other hand, in some cases, these have been apparently normal. In addition to enlargement of the spleen, the mesenteric glands have also been found swollen, and organisms have been found in them. By these facts, the idea of a toxic influence acting on the nerve centres has been suggested. Confirmation of such a view is afforded by the fact that cases of acute multiple neuritis, certainly due to a toxæmia, may run a similar course, and that indications of such commencing neuritis may actually be found in this disease. Such a view does not, of course, exclude a central process; and it is possible that there may be gradations between the pure form of this disease, and cases of acute multiple neuritis of ascending course. All these facts suggest that the virus is most likely a product of the preceding growth of organisms, at least in some cases, and if this is so, it is natural that the organisms themselves should not be found.

Symptoms.—General malaise, pains in the head and back, and tingling in the extremities have been noticed, in some cases, as premonitory symptoms. The first symptom is usually weakness of the legs; this rapidly increases, so that the power of standing is lost, and it goes on to complete paralysis, with relaxation of the muscles. The trunk muscles become similarly affected, and then the weakness invades the arms, and the paralysis of these limbs may become absolute, like that of the legs. The diaphragm and the neck muscles are next affected, difficulty in swallowing comes on, there is dyspnœa, and the cardiac centre may be involved. In rare cases there is evidence of paralysis of the ocular muscles. There may be some blunting of sensibility in the extremities, but no definite loss of sensation. At first, reflex action is lost in the affected limbs, but the muscles present no signs of wasting, or of change in electrical irritability. The sphincters also escape in the vast majority of cases; the cerebral functions are not, as a rule, involved, although in some cases there may be mental dulness or slight delirium. There is no elevation of temperature as a rule. Profuse sweating has been known and noted, and enlargement of the spleen is very frequent. In some cases of irregular course, changes have been found in the electric irritability of muscles. In such cases, it is probable the peripheral nerves were affected. The disease varies in its rate of progress; death usually results from respiratory or cardiac paralysis, and in a large proportion of cases the fatal result occurs within a week. On the other hand, it may only attain its height at the end of two, three, or even four weeks. In some cases, apparently of this disease, paralysis of the limbs has not been complete. In cases that have recovered, recovery was usually slow, and, as a rule, its order is opposite to that of invasion.

Diagnosis.—The disease is recognised by its rapid development, by the presence of paralysis with relaxation of the muscles, loss of reflex action without considerable pain, the absence of loss of sensation or impaired nutrition, and an unchanged electrical irritability of nerve and muscles. The diagnosis from general ascending myelitis rests on the involvement of all the functions of the cord in the latter inflammation. From acute atrophic paralysis it is distinguished by the absence of electrical changes in the muscles. From multiple neuritis the distinction may not be always absolute, since the nerves probably suffer as well as the cord in some of the cases.

Prognosis.—The prognosis is one of extreme gravity, the danger to life being in proportion to the interference with respiration and the functions generally of the medulla. Even when the symptoms only reach their height at the end of several weeks, death may occur in the same way as in the more rapid cases. On the other hand, recovery has been known to take place, although the patient has lost all power of movement at the end of the second day. Mental symptoms increase the gravity of the prognosis, as they usually indicate a severe blood change.

Treatment.—A warm bath, or, better, a vapour bath, should be given if the symptoms follow exposure to cold. This should be followed by counter-irritation over the spine, by a long narrow mustard plaster, or even by the actual cautery. The efficacy of drugs is doubtful. Salicylate of soda seems to deserve a trial in cases following exposure to cold. Ergotin has also been given, and in one case in which it was administered the patient rapidly improved, and at the end of a week was well. If swallowing becomes difficult, care must be taken to administer a sufficient amount of nourishment, either by the rectum or by the nasal tube. Several cases have been recorded in persons who have had syphilis in whom the arrest of the disease followed antisyphilitic treatment. In cases which followed rheumatic lesions, especially if there have been any indications of septicæmia, full doses of perchloride of iron offer the best means of treatment.

CAISSON DISEASE.

Syn., Diver's Paralysis.

Divers, and especially those who work in caissons, may become paralysed soon after they return to the surface. Apoplectic attacks and hemiplegia also occur; but paralysis of the legs is by far the most common effect. Miners have been said to suffer also, but this is doubtful. Most of the subjects of the disease have worked at a depth of from 40 to 90 ft. below the surface of water, and under a pressure of two to four atmospheres. Severe degrees of the affection are rare, because the conditions under which they occur can be avoided. At a depth of 90 ft. beneath the surface immunity from severe symptoms, and commonly from all symptoms, is obtained by reducing the periods of work to an hour. The danger is greater in those who have had several previous periods of work on the same day, especially if slight symptoms have been

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experienced after one of these, and it is also greater in those unaccustomed to the conditions. It is not during the exposure to the abnormal conditions that the symptoms come on, but after the return to the normal atmospheric pressure. The onset is usually preceded by other symptoms, especially pains in the ears and in the joints. The larger joints are those chiefly affected, and very rarely there is also swelling in them. Paraplegia or hemiplegia may occur, the former far more commonly. The onset is sudden, the legs feel heavy, and are found to be weak. Sensation is often lost, and in all severe cases the sphincters are affected. The arms are seldom involved. If the paralysis is incomplete, power may return in the course of a few days, but in severe cases the palsy usually lasts for weeks, or even months, and may be permanent. Death may occur at the end of a week or more, in the same way as in acute myelitis. When the paralysis is hemiplegic, it is usually moderate and transient, unless the symptoms are very severe, with sudden loss of consciousness and deep coma, in which case death usually occurs in the course of a few hours.

Pathology.—The most probable explanation of these cases ascribes them to the escape from the blood of gases, with which it has become charged during the exposure to the high pressure. Gas escaping from the blood within the body, as it apparently sometimes does, is no doubt quickly reabsorbed, but must exert a pressure capable of arresting the functions of the structures of the nervous system. If abundant, it may conceivably rupture these.

Subsequent escape of gas has been proved to result from diminution of atmospheric pressure. It was once thought that the symptoms were due to local hæmorrhages, but extravasations are seldom met with, even as the results of experiment. Leyden found, in a characteristic case of paraplegia, small irregular fissures in the mid-dorsal region, chiefly within the posterior and hinder parts of the lateral column. The fissures were filled with round cells, but contained no blood corpuscles. The only explanation which is satisfactory is, that they are produced by the sudden escape of gas, and are afterwards occupied by round cells. The physical firmness of the cord is far less in the dorsal region than elsewhere, and probably this is the reason why this region suffers structural damage more readily than other parts. Such escape of gas may readily occur in the brain, even to a fatal degree, and leave no traces, unless it occurs so rapidly as to cause laceration. Some other anomalous features, such as the transient character of the symptoms, are also explained by the peculiar nature of the lesion. Hæmorrhage may be regarded as an accidental consequence. The fact that the spinal cord suffers more than other organs may be partly due to the plexuses through which the blood can return only slowly to the lungs, where the excess of gas is gradually given off. It is certain that in all cases of exposure to a high degree of pressure, the transition to the normal should be made gradually. When developed symptoms indicate that a definite lesion of the cord has occurred, the further treatment must be conducted on the same principles as in acute myelitis, the morbid process that has, in fact, been set up.

MYELITIS.

Myelitis, or inflammation of the spinal cord, occurs in several varieties. In acute myelitis the inflammation comes on rapidly, and reaches a considerable degree in a short time. The rapidity with which the symptoms develop is often extreme, and may be almost sudden in the hæmorrhagic form. In subacute myelitis, the onset occupies from two to six weeks. When a longer time elapses before the symptoms reach a considerable degree of intensity, the inflammation is termed chronic, and the cases of very slow course merge into those in which the process is generally regarded as "degeneration" or "sclerosis." No sharp distinction can be drawn between those classes. Other varieties have been named from the anatomical distribution of the morbid change. In one form the grey matter is affected primarily and chiefly—"poliomyelitis"; it is the cause of the common acute wasting palsy of children—"spinal infantile paralysis." When the whole thickness of the cord is affected in a small vertical extent, the myelitis is said to be "transverse." If an extensive area is continuously inflamed, the myelitis is said to be "diffuse"; if only one small area is affected, it is called "focal"; and when there are several foci scattered through a considerable extent of the cord, it is said to be "disseminated." In rare cases the inflammation is "central," being situated in the tissue around the central canal. When the spinal cord is compressed, it is also inflamed—"pressure myelitis." A further distinction has been made between myelitis as it affects the nerve elements—"parenchymatous myelitis," and myelitis which begins in the connective tissue, the affection of the nervous tissue being secondary—"interstitial myelitis." The latter is the common form; the former is chiefly due to toxic agents.

Etiology.—Little is known of the influence of hereditary tendency: it seems to imply a disposition to inflammation, generally it is a disposition to affections of the nervous system. The disease occurs at all ages; but inflammation of the grey matter, "poliomyelitis," as a rule, affects children. Males suffer more frequently than females, partly from greater exposure to the exciting causes—injuries of various kinds, even concussion, and also exposure to cold. Concussion often causes hæmorrhages, around which inflammation may develop and spread. Violent action of the muscles attached to the spinal column has sometimes appeared to excite inflammation in the adjacent part of the cord. The cause of this is uncertain; it may be partly compression of the vessels. Another exciting cause is exposure to cold, especially to wet cold; but it is probable that cold usually acts through the mechanism of a morbid blood state, which it in some way induces, and which varies in its nature and effects according to individual predisposition. Over-exertion is another cause, and is especially effective when combined with insufficient food and exposure to cold.

The sudden suppression of the menses, and of other habitual discharges, has also been supposed to cause the disease. In most instances of this kind there has been simultaneous exposure to cold, and to this possibly the suppression and the myelitis may both have been due. Functional over-action of the cord may cause inflammation in the part which is thus over-stimulated.

Toxic blood states constitute another important cause. They are most varied in origin. Myelitis occurs in a very obstinate form in some cases of gout, and this may be the mechanism by which exposure to cold is effective. It occurs in the course of, or after, many acute diseases, especially measles and variola; and an acute form is frequent in the subjects of syphilis. It is perhaps sometimes due to the organisms of the acute disease, but more often to a toxine they produce. In functional over-action the related disturbance may attain an inflammatory degree.

Many other chronic toxæmic states, as chronic blood states and constitutional alcoholism and metallic poisoning, are causes of both acute and chronic myelitis.

Morbid anatomy.—In recent acute inflammation of the cord the pia mater is usually red, and the cord itself may be swollen. The distinction between white and grey matter is indistinct or lost. In acute inflammation the affected part may be diffluent, probably because post-mortem softening occurs more readily at an inflamed part. When the vascular disturbance is considerable, the condition is termed *red* softening. If much blood is effused into the tissue, it is called hæmorrhagic myelitis. After the inflammation has existed for some time, the colour becomes lighter, giving rise to *yellow* softening; and in cases in which there is little extravasation of blood, but much breaking up of nerve elements, the softening is known as *white.* If the inflammation is very limited in extent, cavities may be formed within the cord.

The microscopical appearances vary much, according to the duration and form of the inflammation. Products of degeneration of the fibres—granules, masses of myeline, the peculiar bodies termed corpora amylacea, numerous red blood discs, leucocyte-like corpuscles, and, after the first week, distinct cells of various shapes, and fragments of axis cylinders, are the most common objects seen on examination. It is impossible, on account of the softened state of the tissue, to learn much of the condition of the parts in which the inflammation is intense. The products of degeneration are best seen in the fresh state in sections mounted in glycerin. The fragments of myeline occupy the position of the nerve fibres from which they have come, or are aggregated into masses. In sections prepared in the ordinary way, the most conspicuous changes are the dilatation of vessels and the accumulation of leucocytes and other cell elements in the tissue. In the white substance, the large vessels are conspicuous by their enlargement, and in the grey substance the dilated arteries and capillaries may occupy a considerable part of the tissue. The walls of the smaller arteries are encrusted with leucocytes, and outside this thickened wall is an enormously distended perivascular sheath. In transverse section this may have the appearance of a very much thickened vessel-wall. Red blood corpuscles distend the vessels, and are sometimes aggregated in small extravasations, the result of the rupture of vessels. The grey substance is densely set with round corpuscles, staining deeply, while the intervening substance is much more granular than in health. The nerve cells are swollen and granular, often containing globules apparently fatty; the cell processes appear shrivelled or lost. In some cases, however, especially those of disseminated interstitial myelitis of moderate degree, the nerve cells appear little changed.

In the white substance, sections show a great increase in the interstitial tissue, either uniform or greatest in the neighbourhood of vessels. The so-called spider cells, or cells of Deiters, are often conspicuous objects. The changes in the nerve fibres are various. The slightest change is that which is manifested by its staining more readily than usual. There is also irregular swelling of the axis cylinder. These intense degenerative changes cause the breaking up of the myeline, and may involve the destruction of the fibre.

The relative amount of change in the nerve elements and in the interstitial tissue varies very much, and its distribution is variable. In pressure myelitis, the whole thickness of the cord is uniformly affected. In transverse myelitis, all the structures at the affected level may be destroyed, and only products of degeneration and inflammation may be visible. In slighter cases the alteration is seldom uniform, while in meningomyelitis the periphery of the cord is most affected, and in old cases the thickened pia may send dense tracts of connective tissue into the interior of the cord. In disseminated myelitis the foci of inflammation may exist through a wide extent, and appear on the surface as reddish grey areas, less sharply outlined than those of disseminated sclerosis. At first, vascular changes are the more conspicuous, but afterwards the evidence of destruction of nerve elements characterises these foci, and ultimately a condition of irregular focal sclerosis remains. In central myelitis there is a great increase in the nuclei about the central canal, and vascular changes in the adjacent commissure, but there may also be evidence of diffuse myelitis. Little is known of the morbid appearances in the parenchymatous forms, except that in diphtheritic and other toxic paralyses, as well as in a variety of poliomyelitis, the nerves present granular change, and the interstitial substance is but slightly affected. The nerve roots coming from an inflamed part are usually damaged, and degenerative and sclerotic changes may descend the motor nerves. From the foci of inflammation, ascending and descending degenerations pass along the tracts that have been already mentioned as those which such degenerations effect, and definitely inflammatory changes may be traced in certain tracts for a short distance above and below the chief lesion.

If the patient survive, the new interstitial tissue undergoes changes which give it a fibroid aspect, although for some time cell forms may preponderate in it. We know very little of the nature of the process which occurs in the cases which recover slowly. An absolute motor palsy of twelve months' duration must depend on changes that involve an absolute interruption of the fibres, and yet in such a case some return of power may occur. It has not yet been demonstrated that such an effect is produced by a growth of new fibres, although in the lower animals such a growth has been proved to occur.

Pathology.—Very little is known of the actual mechanism by which acute myelitis is produced, or the nature of the causes to which it is due. It is a question of some interest whether acute inflammation of the cord always begins as such, or whether it is sometimes set up by vascular obstruction, such as thrombosis in a minute vessel. Experiments on animals show, however, that the lumbar enlargement is damaged with special readiness. This suggests that the proclivity of this part to disease is due to its inherent susceptibility.

At present the questions of chief interest are, the extent to which the disease is due to a morbid blood state, the probable nature of such a cause, and the mechanism by which it acts. The chief fact likely to throw light on this subject is the occurrence of myelitis in the acute specific diseases, and the evidence of an influence acting throughout the system afforded by the wide extent of the disseminated form, and its occasional association with disease elsewhere, such as optic neuritis. Of the pathology of simple transverse myelitis we have only the fact that it frequently seems to follow exposure to cold. If the cause of the myelitis is a persistent influence, such as a toxic agent, it is easy to understand that the local influence of the lesion itself will co-operate with that of its cause, and induce the progressive tendency that is characteristic of this form.

Symptoms.—The characteristic symptoms of myelitis depend on interference with the functions of the cord. These are sometimes preceded, more often accompanied, by the usual symptoms which attend inflammation of other internal organs—malaise, shivering, headache, depression, loss of appetite, fever. When due to cold, and commencing some days after the exposure, general rheumatic pains may occur in the interval. The most obtrusive spinal symptom is motor weakness; but this may be preceded by numbness, tingling, or burning sensations. Occasionally there are pains in the joints at the onset, and spasmodic twitchings or painful cramps in the limbs. In rare cases—and this is especially true of children—there is a general convulsion.

The motor paralysis usually comes on rapidly, and in the course of a few hours has reached a considerable degree. Occasionally the onset occupies only a few minutes, and in such cases there is probably hæmorrhage in addition to inflammation. Now and then the onset is in the night during sleep. On the other hand, the onset may occupy several days, or even a week. In still other instances, the onset is by a series of sudden attacks of weakness, occurring at intervals.

When the paralysis has reached its height, it is usually complete. Sometimes a little power of movement remains in the limbs, and the distribution of the paralysis depends on the position and the extent of the disease. In most cases the legs and lower part of the trunk only are affected. When the disease, however, is in the cervical region, the arms as well as the legs are involved, and in such cases the arms are frequently paralysed before the legs. If the lesion is extensive in the cervical region, the damage to the grey matter and root fibres causes atrophic palsy in the arms; while the legs are the seat of simple spastic paralysis. Myelitis above this level may, by involving the regions of the phrenic nerve, cause paralysis of the diaphragm, and endanger life by the great degree of interference with respiration which this necessarily involves. In such cases disseminated inflammation may spread into the medulla, and the heart's action also be interfered with.

Sensation is frequently impaired as well as motion. In severe cases, it is lost up to the level of the lesion. In slighter cases the loss may be partial. Occasionally there is general hyperæsthesia in the limb at the onset. At the level of the lesion there is usually a zone of hyperæsthesia, probably corresponding to the merely irritated part of the cord above the lesion. Corresponding to this zone, there is often a sense of painful constriction—a "girdle pain." This is sometimes a very early symptom. When pain persists in the legs, it is often less when these are flexed.

The state of reflex action varies, and depends upon the position of the lesion. An acute lesion in any part of the cord may cause at first inhibitory loss of reflex action in the parts below; but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours, and in time becomes exaggerated. Frequently there is no initial depression. If the disease involves the lumbar enlargement, all forms of reflex action are lost. The superficial reflexes should always be examined at various levels, and the greater impairment of these on one side may reveal clearly the unequal distribution of the lesion, and its vertical extent.

The muscles of the leg are sometimes at first flabby and toneless; but if the lesion is above the lumbar enlargement, this condition soon passes off, and the muscles regain their tone. If, however, the myelitis involve the lumbar enlargement in considerable degree, the muscles of the legs remain flaccid, the feet fall into an extended position, the muscles waste rapidly, and often present the reaction of degeneration.

The sphincters are usually affected from the first, and they often afford the earliest indication of the commencing disease. There is usually at first retention of urine, and afterwards incontinence, if the disease is above the lumbar region. If the lumbar centre is diseased, there is persistent incontinence without previous retention. In cases of partial myelitis, as of the anterior cornua, the sphincters are frequently unaffected. The urine is often alkaline in reaction, and in all cases of retention, decomposition in the bladder occurs, and increases the alkalinity. Cystitis often follows, so that the urine contains pus.

The temperature of the paralysed limbs is usually at first raised one or two degrees, but it afterwards falls, and remains a little lower than that elsewhere. The skin is usually dry, its nutrition frequently suffers, and bedsores result. These are sometimes due to long-continued pressure and neglect, but they may occur early and come on rapidly when the disease affects the lumbar enlargement, either primarily or secondarily. Now and then there is effusion into the knee-joints, and occasionally the tendency to sloughing is so intense that the lesions appear to be spontaneous. Such trophic disturbances sometimes occur in the cellular tissue of the lower part of the body, especially near the bladder and rectum. It is probable that the derangement of the trophic influence of the cord facilitates the occurrence of cystitis. Ulceration of the bladder may be thus produced, or a small abscess form in its wall, which may rupture into the peritoneal cavity and produce peritonitis. A vesico-vaginal fistula may be produced in the same way.

The pupil may be affected when the disease is in the cervical region. In rare cases, optic neuritis has been observed, without any intracranial complication to cause it. Such optic neuritis is probably an associated lesion, the result of the cause of the myelitis, which in such cases is probably a toxic blood state. Most of the cases in which this has occurred have been instances of disseminated myelitis.

In some cases the degree attained by the symptoms at their onset may not be exceeded. In others a slow increase ensues in the course of a few days. The upward extension may be steadily progressive, involving arms first, and respiratory muscles later, or it may occur in successive separate attacks. Sometimes inflammation beginning in the dorsal region may slowly descend into the lumbar enlargement: the extension may be by the grey matter, or by the posterior region. In the former case there is rapid muscular wasting, with loss of faradic irritability; in the latter the muscles retain their nutrition and irritability, but sensation becomes lost.

The constitutional symptoms which have been mentioned as attending the onset vary greatly in their severity and course. In most cases they reach their height in the second or third day after the onset of severe symptoms. In less acute cases, in which foci of inflammation develop progressively with spreading palsy, irregular pyrexia may go on as long as the disease is increasing, and may be renewed at any subsequent relapse.

If the patient survive, the course of the symptoms varies. Usually sensation is recovered after a few weeks or months, while motor power remains absent for a much longer period. The excessive reflex action may lead to the development of spasm in the legs, and this is specially related to the increase of muscle reflex action consequent on the descending degeneration of the pyramidal fibres. The occurrence of this spasm is not incompatible with the recovery of some voluntary power. The muscles in these cases are often well nourished, but spasm may co-exist with a moderate degree of wasting in the muscles. Flexor spasm, in some cases, after a time takes the place of extensor spasm. It may be so great that the knees are against the abdomen and the heels against the nates. This spasm occurs chiefly in cases in which the paralysis is absolute, and the organic disease of the cord so severe and of such duration as to make recovery impossible.

Death may occur early in the disease from respiratory paralysis, or from extension of the inflammation into the medulla oblongata. Later it may be due to various effects of trophic disturbance, to blood states induced by bedsores, or to kidney disease resulting from cystitis. Improvement, when it occurs, is usually slow, but recovery may be complete in cases of moderate severity. When, however, there is much damage to the cord, recovery is usually imperfect. It is rarely that no improvement occurs, but in some cases improvement is followed by a relapse which leaves the patient worse than he was after the first attack. This may occur repeatedly, each relapse being the expression of a fresh extension of the inflammation in the cord. In many cases, especially of simple transverse myelitis, improvement once commenced goes on, and there is no tendency to recurrence.

The different pathological forms of myelitis are attended by certain differences in the symptoms. The description already given applies more particularly to transverse myelitis. In focal myelitis the symptoms depend upon the position in the cord of the focus of inflammation. In disseminated myelitis, in which there are several foci of inflammation, the onset is often subacute, and constitutional symptoms are frequently absent. In many cases the several foci develop successively, not simultaneously, and then we have a corresponding succession of symptoms. In the rare form of diffuse central myelitis there is usually rapid loss of power, of sensation, and of reflex action, considerable elevation of temperature, speedy trophic disturbance, and often death at the end of two or three days. Hæmorrhagic myelitis is scarcely a special form, since any acute inflammation of the cord may be attended by extravasation of blood. Parenchymatous myelitis is known only in a few forms, and we have much yet to learn regarding it. In diphtheritic paralysis there is an acute degenerative change in the nerve elements, especially the nerve cells, which must be regarded as essentially a lesion of this form. In some cases also of poliomyelitis, the motor nerve cells suffer primarily. Other symptoms that are of importance as suggesting this variety are those of acute ataxy, clearly dependent on an affection of the cord.

Diagnosis.—Myelitis is recognised by the rapid onset of symptoms indicating structural disease of the cord. Among those, the failure of power is the most significant, although subjective sensations that accompany it are often more obtrusive at the onset. No one of the early symptoms is more important for its definite significance than retention of urine, and its importance is increased by the fact that it may precede all symptoms in the legs. The diagnosis may be aided by the presence of the general symptoms of inflammation, although constitutional disturbance is as likely to mislead as it is to suggest the local affection. The position of the myelitis will be indicated by the symptoms. In ascertaining the state of the lower dorsal region, the trunk and cremasteric reflexes are especially important. The state of the muscles as regards nutrition and electrical irritability is very important, but is only useful several days after the onset. The distinction from simple poliomyelitis depends on the fact that the symptoms are not purely motor, and it is only when the myelitis is cervical or lumbar that the diagnosis in reference to this disease is a matter of difficulty.

The distinction of the other varieties of myelitis rests on the special features already described. That of the disseminated form rests on the irregular distribution of the symptoms, and it is doubtful whether central myelitis can be diagnosed during life. Acute myelitis in syphilitic subjects, even if due to syphilis, presents no distinguishing features, nor is its course influenced by antisyphilitic treatment.

If transverse myelitis has been diagnosed, it should always be asked, "Is it primary, or secondary to some other process?" of which the most common is compression of the cord. The spinal column should be carefully examined in all cases, not merely once, but again and again. Myelitis in a cancerous patient should always suggest secondary growth in the spine. The diagnosis from other lesions of the cord is chiefly by the mode of onset. In hæmorrhage, the symptoms develop in a few minutes. When acute myelitis ascends the cord, so that the legs, the muscles of the trunk, and the arms are successively paralysed, its course resembles that of acute ascending paralysis, or Landry's paralysis. The most important distinction is that in the latter sensation is unaffected, and there is no tendency to trophic disturbance or muscular wasting, or change in electrical irritability. In meningitis, the symptoms of irritation are prominent, and in meningeal hæmorrhage there is severe pain in the back, and acute irritation of the nerve roots. The distinction from multiple neuritis is important chiefly in cases of poliomyelitis, but a difficulty may exist when parenchymatous myelitis affects the structures on which co-ordination depends, and produces the condition of myelitic ataxy. Myelitis is far from rare in patients of the age and sex in which hysteria especially prevails, and many cases are mistaken for hysterical paraplegia. The mistake occurs especially in cases of transverse dorsal myelitis, where there is no wasting of the leg muscles. If one leg is lifted from the bed, and the other moves with it, owing to the rigid extensor spasm, organic disease is certain. Hysterical contracture never fixes the legs to the pelvis so as to permit this effect. In hysterical paraplegia there may be retention of urine, there is no incontinence. Girdle pain, incontinence of fæces, trophic changes in the skin, are conclusive symptoms of organic disease.

Prognosis.—The primary danger to life in myelitis depends on the risk of respiratory palsy, and hence on the region of the cord in which the disease is situated. A disposition to spread is always of grave significance, and therefore the prognosis is worse in disseminated than in simple myelitis. The risk is always great when the cervical cord is diseased, and any indications of the development of foci in the medulla oblongata are of grave significance. Acute trophic disturbances also constitute a danger, and hence bedsores, if forming early and rapidly, are of unfavourable omen. The occurrence of cystitis increases very much the gravity of the case. The prospect of the recovery of power arises when the disease has become stationary. It depends on the intensity of the disease, its vertical extent, and the early occurrence of symptoms of improvement. It is influenced by the cause of the myelitis, and it is better when there is evidence that the disease is confined to the dorsal region of the cord. The development of increased myotatic irritability shows that improvement will not speedily occur, but even rigidity in the shape of spastic paraplegia does not lessen materially the prospect of some recovery. The fact of preceding syphilis should have little or no influence on the prognosis in a case of simple myelitis.

Treatment.—If a case comes under observation at the earliest period, and the symptoms are clearly due to exposure to cold, a hot bath, followed by free diaphoresis and counter-irritation, should be employed. If considerable paralysis shows that the process of inflammation is fully developed, rest is of paramount importance. In regard to position, it is most undesirable that the spine should be the lowest part of the body. A plank back-rest will be found useful in securing comfortable rest on the side, and the prone position should be adopted at times as a change from the lateral. The removal of blood by leeches or wet cupping from the skin of the back is an old measure, and may have some influence on the circulation of the cord. Dry cupping may also be employed, or hot fomentations, hot-water bags, or a mustard plaster. The application of cold to the spine has also been recommended, but probably the application of warmth is safer, more effectual, and more pleasant. In other respects the treatment must be determined by the nature of the process rather than by the character of the organ in which it occurs. Nutritious, unstimulating diet, aperients, and diuretics are desirable in all cases. A free purgative may be given if there is constipation. It is difficult to ascertain the influence of the drugs which are supposed to exert a special influence on myelitis. Ergot was recommended by Brown-Séquard, chiefly on theoretical grounds. In rare cases it has seemed to do good. Belladonna also has been recommended, and mercury by the mouth or by inunction. The effect of the last-mentioned is less distinct in myelitis than it is in many cases of meningitis. Iodide of potassium seems to be no more efficacious than mercury. In the general management of a case of myelitis, two points are important—(1) to avoid bedsores, and (2) to relieve the retention of urine. The skin should be carefully watched, especially if there is incontinence of urine, and boracic or salicylic absorbent wool should be applied, and changed as often as it becomes saturated. Should offensive sores form, a quantity of picked oakum placed outside the immediate dressing is cheap and effective as a deodoriser and dressing. If the bladder is left full, and the urine allowed to dribble away, inflammation is sure to be set up, and probably also pyelonephritis. If cystitis occurs, antiseptic washes must be used, to lessen as far as possible decomposition of the urine.

When the disease of the cord has become stationary, a more tonic treatment may be adopted; iron, quinine, and arsenic being administered, but strychnine only in small doses, if there is excess of reflex action. The limbs may be rubbed, electricity may be used, in order to maintain as far as possible the nutrition of any muscles of which the nerves have undergone degeneration. In cases of dorsal myelitis, in which the legs are well nourished, electricity is useless, and may tend to increase reflex over-action. Attention should in all cases be paid to the position of the limbs during the stage of helplessness, so as to avoid the development of contractions.

HÆMORRHAGE.

Primary hæmorrhage into the spinal cord is a very rare condition. Hæmorrhage may accompany myelitis, and in such a case the mistake may be easily made of regarding the hæmorrhage as primary. When inflammation results from hæmorrhage, and when indications of myelitis are found around the clot, it may be impossible to say whether these are primary or secondary. The exclusive view of some writers, that primary non-traumatic hæmorrhage into the spinal cord never occurs, is not warranted.

Etiology.—Compared with the frequency of hæmorrhage into the brain, hæmorrhage into the spinal cord is extremely rare. This difference probably depends on the long, tortuous course of the arteries of the cord, whereby the vessels are preserved from the high pressure to which the cerebral arteries are so prone. Hæmorrhage may occur at any age, is more common in males than in females, and is more common during the first half of adult life. Many cases occur in young adults, apart from injury or obvious exciting cause. It has been met with as a consequence of the hæmorrhagic tendency; but of immediate causes, injury, especially falls, is the most frequent cause. Over-exertion and exposure have, in rare cases, preceded the onset. Chronic alcoholism and sexual excess have been supposed to predispose to it. Minute extravasations, termed accessory hæmorrhages, have often been found after death from conditions which give rise to venous congestion; but they cause no symptoms, and are probably produced during the last moments of life.

Morbid anatomy.—Small extravasations, referred to as occurring in the asphyxial conditions, may occupy the perivascular sheath, or they may extend between the nerve elements. The larger non-traumatic hæmorrhages, which cause symptoms, always begin in the grey substance, and are often confined to it. The effusion forms a cavity in the cord, and the cord itself is enlarged at the seat of the hæmorrhage. This may be visible externally as a dark swelling. Very rarely the hæmorrhage tears the cord, and blood (usually only in small quantities) escapes into the membranes. The tissue adjacent to the clot is usually broken down, stained and softened, and inflammatory changes may be recognised in it with the microscope. Several extravasations may coexist in the same part of the cord. Besides this focal hæmorrhage, there may be an infiltration of the grey matter, with punctiform extravasations, which may appear to be a single hæmorrhage. The tissue between these extravasations is broken down, and it is probable that this form is always secondary to myelitis. A growth into which hæmorrhage occurs is usually a glioma. The cavities of the cord into which blood may escape are sometimes of considerable vertical extent, and this condition favours the occurrence and extension of the condition.

Symptoms.—Trifling sensory disturbance may precede the paralytic phenomena, but the actual onset is always sudden, and the symptoms attain a considerable degree in the course of a few minutes. Sometimes these have been slow, and increase during one or two hours, and occasionally there has been transient loss of consciousness, without any cerebral lesion. The suddenness of the onset is the characteristic of the disease. The symptoms which thus develop vary according to the seat and extent of the hæmorrhage. There is usually complete motor and sensory paralysis up to the level of the lesion, and loss of control over the sphincters. Pain may or may not accompany the sudden palsy. This pain may precede the palsy, even for half an hour or an hour, and is no doubt a result of the first slight extravasation. When there is initial mesial or bilateral pain, the hæmorrhage probably occurs in the central region of the grey matter, and the pain may be due to the irritation of the fibres of the posterior commissure. The spinal column may be tender opposite the affected spot. Usually the paralysed muscles are relaxed; sometimes they are the seat of clonic contractions. The state of the reflex action varies according to the seat of the disease. If it is at first abolished, it quickly returns to the legs, unless the hæmorrhage is in the lumbar enlargement. Vasomotor and trophic changes in the skin are common, and often intense. The temperature is normal at the outset, but generally rises in the course of a few days, from the secondary inflammation of the cord. The palsy developed at the onset usually continues for a week or ten days; and occasional symptoms of an ascending or descending myelitis may come on, the former of which may cause death by interference with the muscles of respiration. The occurrence of descending inflammation may be shown by the loss of reflex action, and by the failure of electrical irritability in the muscles. As a rule, the symptoms pass into a chronic stage, improvement generally being slow. Some lasting loss of power remains in most cases, often associated with permanent muscular wasting. Occasionally there is rapid recovery up to a certain point, caused, no doubt, by the contraction of the clot, which has pressed upon, but not destroyed, certain structures of the spinal cord.

Diagnosis and prognosis.—The actual sudden onset is one of the most important signs in distinguishing this disease from others. Even this may not distinguish it from hæmorrhagic myelitis, and meningeal hæmorrhage. The former is distinguished by the existence of slight symptoms before the attack; and the distinguishing points from meningeal hæmorrhage will be found under the description of that disease. The prognosis is better when the disease is in the dorsal region than when in the enlargements. It is better if sensation returns in the course of a few days. But early trophic changes always render the prognosis worse. After the onset, the forecast must be guided by the same considerations which affect the prognosis in acute myelitis.

Treatment.—The measures requisite are few, simple, but all-important. Absolute rest in the prone position, ice along the spine, and full doses of ergot or ergotin, comprise the most effective means of treatment. The disease is one of those in which most of the good that can be done by treatment rests with those in whose hands the patient is immediately after the onset.

COMPRESSION.

There are various causes of compression of the spinal cord, but the importance of compression arises from the fact that inflammation is almost always excited at the seat of the pressure, and interference with function occurs from the resulting myelitis.

Etiology.—The chief causes are disease of the bones, especially caries, growths in the spine, aneurysm, eroding bones, growths in the membranes, thickening of the dura mater.

Pathology.—The cord is usually narrowed at the point of pressure. Sometimes this narrowing is extreme; on the other hand, it may be very slight. At the point of pressure the cord is usually grey in tint, its consistence is lessened in early cases, the change in colour and consistence being due to the inflammation of the substance of the cord, which results from pressure. The inflammation consists in a general increase in the interstitial tissue, the nerve elements undergo degeneration, masses of myeline, granule corpuscles, and corpora amylacea are visible with the microscope. Many nerve fibres persist, however, with perhaps a narrowed sheath of myeline, but in extreme cases all the fibres seem to be destroyed at the point of chief compression. The walls of the small vessels are often thickened by spindle cells, so that the lumen is narrowed, and this, of course, must further interfere with the nutrition of the cord. The signs of inflammation gradually lessen above and below the compressed part. The nerve roots may suffer at the point of compression, and may even be reduced to fibrous shreds.

Symptoms.—The symptoms depend upon interference with the function of the nerve roots at the level of the morbid process, and interference with the functions of the cord itself. Of the root symptoms, the most constant is pain, usually sharp, and sometimes accompanied by tender points. The pain may be intermittent or constant, and with it there is usually hyperæsthesia of the skin, and after a time anæsthesia may develop in irregular areas, in spite of the persistence of pain—the condition termed *anæsthesia dolorosa.* Irritation of the motor nerve roots may cause muscular weakness, accompanied by wasting, and sometimes also painful contracture of the muscles. To these, local root symptoms are added; after a time, the symptoms of interference with the function of the cord itself. There is weakness in the parts below the lesion, there is marked increase in the superficial reflex action, the myotatic irritability is increased, and foot clonus is nearly always present. There is often formication and tingling, but there may be no impairment of sensibility, even when there is complete motor palsy. In many cases, however, there is some sensory loss, in not a few it is considerable, and may even be complete. The course of the symptoms varies according to the nature of their cause. If compression ceases to increase, the cord may recover its conducting function, although it remains narrowed. Sensation is often regained in the legs when motor power remains in abeyance. In some cases, especially where a considerable myelitis has been set up, the motor path may recover, but the posterior columns remain affected, so that power returns, but with impaired co-ordination, secondary ataxy resulting.

Diagnosis.—The diagnosis rests on the presence of the root and the cord symptoms already described. The chief disease from which it has to be distinguished is a subacute transverse myelitis. The chief distinction depends upon the presence of symptoms indicating irritation of the nerve roots. The distinction from a growth within the spinal cord rests chiefly on the early occurrence of impairment of the central or conducting functions of the cord, and also on the partial distribution and often slower course of the symptoms. The diagnosis of the cause of compression is often much more difficult than that of its occurrence. If the patient is in the first half of life, especially if there is a tuberculous tendency, caries is naturally suggested. Slightness of root symptoms is also in favour of caries or of tumour of the spinal cord. Root symptoms, extending over a considerable vertical area, suggest pachymeningitis.

Prognosis and treatment.—These, of course, depend on the cause, and the amenability of this cause to treatment. As a rule, the more rapidly the cord symptoms develop, the more chance there is of improvement, because in such cases inflammation takes the greater share in their production.

TUMOURS.

Morbid growths within the canal may spring from the membranes, or grow in the substance of the cord itself.

Etiology.—There is nothing to be said in reference to the etiology of growths of the spinal cord that is not true of growths elsewhere. Fatty growths outside the dura mater occur in early life; malignant tumours develop late. Tumours within the dural sheath, myxomata, are chiefly met with in early life; tuberculous growths occur in childhood; lipomata are congenital. Males are more prone to suffer than females. Of the diathetic conditions, syphilis and tubercle are the only two which are effective in causing growths which commence within the spinal canal. Parasitic tumours occur, and a few rare growths appear to be congenital in their origin, the result of the abnormal development of germinal tissue; while the more common gliomata within the cord arise from embryonal tissue which has undergone arrest in its development. Of the causes of other forms of tumour we know practically nothing.

Morbid anatomy.—The growths may be outside the dura mater, inside the dura mater, or in the substance of the cord. The forms of extradural tumours are lipoma, and parasitic tumours, chiefly echinococci; but all are rare. Growths also occur springing from bones, but these have been already considered. The tumours which begin within the dural sheath are chiefly syphilomata, sarcomata, and myxomata, sometimes containing cysts or brain sand. The tuberculous and parasitic tumours are rare. Fatty tumours have been found, and may contain muscular fibres. They are congenital, and sometimes associated with spina bifida. Neuromata occur in the nerve roots, and may compress the cord.

The growths within the substance of the cord are more diverse in character. Syphiloma and glioma are the most common. Sarcomata, myxomata, and tuberculous growths also occur. Cysticercus has been met with, and some growths have a compound character, such as myxo-sarcoma, gliosarcoma, and fibrosarcoma. Sarcomata and gliomata are sometimes very vascular, and have been termed angiosarcoma and angio-glioma. Extradural growths are always single; those within the dura mater are often single, but sometimes multiple, or the growth may be diffuse. Neuromata or sarcomata on the nerve roots are often multiple, and tumours within the cord are usually single. The size varies from that of a pea to a width of 1, and a length of 2 in. Those developing from the pia mater are usually smaller than those which spring from the dura. The compression produced by tumours within the dura mater is always greater than that of tumours outside the membrane, and the amount of pressure is proportioned to the size of the tumour and its consistence. At the compressed part the cord is narrowed and softened, and usually indented or flattened. The softening depends on the inflammation, and secondary inflammation may extend down the cord below the growth, or for a short distance above it. The usual secondary degenerations are commonly conspicuous.

The growths which involve the cauda equina often attain a larger size than those that occur higher up, because that part of the vertebral canal is large, and nerve roots occupy but a small part of it. Most tumours in this situation are sarcomata, but it is not an uncommon seat of syphilitic growths. Multiple tumours outside the cord are sometimes very numerous. They are usually sarcomata, and spring from the membranes, or sheaths of the nerve roots. They vary in size. Tumours within the spinal cord are usually small, seldom exceeding half an inch in diameter. But sometimes their vertical extent considerably exceeds their transverse diameter. This is especially true of gliomata, which have been known to reach from the medulla oblongata to the lumbar region. The cord is enlarged at the seat of the tumour, and in some cases this enlargement is very great. If the tumour reaches the surface, the pia and arachnoid may be thickened and may be even adherent to the dura mater. Syphilitic tumours commonly arise in the pia mater, and they both invade and compress the nerve tissue. In all forms of tumour, the central canal may be obliterated by the pressure, and may be slightly dilated above the point at which the pressure exists. In other forms of tumour, cysts occasionally develop, and hæmorrhage may occur into such cysts.

Symptoms.—The symptoms produced by tumours within the spinal canal resemble those that have been described as resulting from growth and caries in the bones of the vertebral column. In the majority of cases pain is prominent throughout the whole course of the disease. It is usually the earliest effect, and may be intense. It often has a peculiar burning character, and is sometimes stabbing or rending. Pain is often felt on one side first, but occasionally it is bilateral. It is generally greatest, according to Horsley, when the tumour compresses the cord from behind forwards, or *vice versâ*. It is sometimes increased by movement, but not often in the intense degree common in tumours of the bone. The root pains in the trunk are often accompanied by a sense of constriction, and tenderness of the spine often exists, but is, on the whole, not common. Occasionally pain is altogether absent.

Muscular spasm is another common symptom. Rigidity of the back may also be present, usually when the disease is at the more mobile parts of the spine. Contractures may develop in the limbs, both in the muscles supplied from the level of the tumour, and in parts supplied from a level in the cord below the growth. Paralysis, of gradual onset, is an almost invariable symptom. Paraplegia is the most common form, but all four limbs may be paralysed by a growth in the cervical region. Usually one leg is affected before the other, and occasionally one arm and leg suffer before those on the other side. The palsy is usually slow in its extension, but in a large number of cases the loss of power comes on in subacute or even acute manner, in consequence of the myelitis excited by the pressure.

Loss of sensation may come on with the loss of motor power, and may be slight or partial at first, tending to become complete as the damage to the cord increases. It corresponds in distribution to the motor palsy, and in a certain number of the cases the cross-symptoms of a unilateral lesion are well marked. Inco-ordination of movement is not a common symptom of spinal tumours, but has been occasionally met with. The condition of the reflexes depends on the position of the tumour. A growth in the lumbar enlargement or the cauda equina abolishes reflex action in the legs, but when the disease is higher up the knee-jerks are increased, ankle clonus is usually present, and muscular spasm and contracture may supervene. Wasting in the muscles is for the most part confined to the region supplied by the damaged nerve roots. Vasomotor disturbance sometimes occurs in the early stages, and sometimes vasomotor œdema is conspicuous. In the later stages bedsores are very common, and often severe. Dilatation or contraction of the pupil is common when a tumour is situated in the lower cervical region.

The course of the symptoms is variable. In some cases the symptoms are steadily progressive from first to last; in others the progress is intermittent. The most important element in the course of the disease is the tendency to a lateral extension of the symptoms, as the several structures of the cord at the same level successively suffer.

The chief difference between tumours of the cord and of the membranes, besides those already mentioned, are that in the latter the root symptoms are more often severe, and the cord symptoms occur later. Extensive muscular atrophy is more common in tumours of the cord than in those of the membranes, except in the case of tumours of the cauda equina, because central tumours may damage the grey matter through a considerable vertical extent, and because myelitis spreads in the grey matter far more frequently when the tumour is in the cord itself than when it is in the membranes. In tumours of the cervical region the interference with movement of the neck, from rigidity of the muscles, is often well marked. Pains are felt in the arms, often associated with muscular atrophy. The excessive reflex action in the legs is very great, and if the tumour is unilateral, hemiplegic weakness, with crossed anæsthesia, may be present. Growths in the dorsal region of the cord usually cause severe radiating pains in the trunk, often with an intensely painful sense of constriction. There is also usually interference with trunk reflexes. Spinal tenderness may be present. Sometimes, when the growth is near the lumbar enlargement, and there is descending myelitis, the knee-jerks may be abolished. The symptoms produced by tumours which involve the lumbar enlargement, vary considerably according to their precise position. The characteristic pains are felt in the legs, and the muscles supplied from the part invaded undergo atrophy and present loss of reflex action. The anæsthesia is often limited in area. In lumbar tumours there is early palsy of the bladder and sphincters. The tendency to bedsores is unusually great.

Growths situated in the cauda equina, not affecting the cord itself, produce symptoms very similar to those due to tumours in the lumbar enlargement, only the symptoms are bilateral from the first in the majority of cases. Trophic disturbances are very frequent, and an affection of the sphincters almost invariable. Cystitis and its consequences readily occur, and may reach a high degree, without any consciousness on the part of the patient.

Multiple tumours may give rise to complex symptoms. Their presence may be indicated by the successive development of focal symptoms, similar in character but different in seat. The duration of the symptoms of tumours of the cord and membranes is extremely variable. In the majority of cases, death ensues at the end of from one to three years from the commencement of the symptoms; death being usually the result of the direct or indirect effect of the tumour. But in syphilis, death is sometimes due to disease elsewhere, and in tubercle, frequently to general pulmonary tuberculosis, and sometimes to meningitis.

Diagnosis.—The evidence of a growth exists in the symptoms of a focal lesion of slow development, and especially in the presence of signs of compression and irritation. No symptom is actually pathognomonic, yet a combination of symptoms and their course are in most cases characteristic. The most important features are the steady progress of the symptoms, and especially their commencement on one side, and their extension to the other. Of causal indications that occasionally assist the diagnosis, the most important are a history of syphilis, the presence of tubercle elsewhere, and, rarely, the existence in other parts of such growths as are known to occur also within the spinal canal, especially neuroma and sarcoma. The differential diagnosis involves the distinction from diseases of the vertebral bones of the membranes and of the cord itself. Caries of the spine, and growths in the bones of the spinal column, produce similar symptoms, but in caries root pains are seldom very severe, and when the growth is in the bone the degree to which pain is increased by movement is very significant. Hypertrophic pachymeningitis affecting the cervical region is the only meningeal disease which closely simulates the symptoms of tumour. But its effects are usually from the first bilateral, and may have considerable vertical extent. But in central tumours the pains in the arms are usually much slighter in this condition, and there is rarely the early and limited impairment of all forms of sensation which results from the damage to the nerve roots in the latter disease. This is also true of syringomyelia.

The affection of the cord itself which most closely simulates the symptoms of tumour is a very chronic transverse myelitis. But it is very rare to get severe and acute radiating pains in focal myelitis, although painful girdle sensation is common. Acute or subacute myelitis can only be confounded with a growth in cases in which inflammation results from pressure, and develops with independent energy and rapidity. The pains produced by tumours of the cauda equina are felt first in the sciatic area, and are thought to be sciatica. But they are commonly from the first bilateral, and bilateral sciatic pain should always suggest central disease. In all cases other symptoms soon become added, and prove that this pain is more than simple neuralgia. It is most important not to permit a mistake in diagnosis by attaching undue importance to evidences of functional derangement, or the gravity of organic symptoms to be underestimated. If the existence of tumour is clear, two questions remain—its seat and its nature. As regards its seat, the considerations which have been adduced in previous pages will sufficiently indicate this. But the nature of the tumour is also of great prognostic importance. The coexistence of a cerebral lesion increases the likelihood of syphilis, but it must be remembered that cerebral and spinal tumours other than syphilitic may coexist. Similarly, tuberculous and scrofulous processes suggest the tuberculous nature of a tumour. Tumours elsewhere always constitute strong evidence of the nature of one in the cord. Such an indication is rarely available. A diagnosis founded on mere frequency of occurrence has necessarily only a low degree of probability.

Prognosis.—Unless the tumour is syphilitic, or can be removed, the prognosis is necessarily grave. In the case of growths outside the cord, the prognosis has been rendered much less gloomy by the progress of surgery.

Treatment.—If the growth is syphilitic, the treatment should be energetic and prompt. Even the influence of a few days may be of extreme importance. Whenever the symptoms suggest a tumour, and syphilis is possible, it is important that suitable treatment should be employed. It can do no harm if the tumour is not syphilitic, and may save the patient's life if it is. In most other cases we can do little more than treat symptoms, relieve pain, watch the state of the bladder, and guard the patient from bedsores. We must remember that sedatives will probably be needed for a long time, and they must therefore be used as sparingly as possible at first. Cocaine may be used in some cases as an aid to the influence of morphine. But many tumours within the spinal canal may be removed, at any rate if they are outside the substance of the cord. But growths within the spinal cord are in a very different position, since the removal of such a tumour would hardly fail to cause a traumatic inflammation, which would damage and perhaps destroy all the elements of the cord at the spot, and would probably be permanent in its effects. In connection with an operation the question sometimes arises, how long should be allowed for the process of exclusion of syphilis by the inefficacy of anti-syphilitic treatment? Longer is needed when the symptoms are of considerable duration than when they are recent; but a month is probably always enough to enable a result to be reached which will justify either further delay on the one hand, or immediate procedure on the other. If it is clear that the growth is not syphilitic, delay in operation can only cause harm.

INFANTILE PARALYSIS.

Syn., Anterior Poliomyelitis; Acute Atrophic Paralysis; Atrophic Spinal Paralysis; Essential Paralysis of Children.

Such are the various names under which the disease is known. The onset of the palsy is often preceded or accompanied by indications of general disturbance, and is the most common form of paralysis in young children. The spinal lesion was first demonstrated by Prévost, soon afterwards by Lockhart Clarke, and by Charcot and Joffroy. The occurrence of similar symptoms in adults was noticed by Vogt and Duchenne.

Etiology.—The disease occurs at all ages, but is ten times as frequent in the first decade as in all the rest of life. Of the cases in early life, three-fifths occur in the first three years of life. The friends often date the disease from birth, but there is no evidence of its intra-uterine development. After childhood, it is most frequent between 10 and 20, and is very rare over 40. The influence of heredity is small, but a family tendency to suffer may now and then be apparent. The disease occurs far more frequently in summer than in winter. It would seem that the heat generally acts as a remote cause, and is only occasionally concerned in the exciting mechanism. That exposure to cold is an occasional cause is certain, and it is usually a general and not a local exposure, often immediately preceded by exposure to heat. Over-exertion in walking seems occasionally to aid in exciting the disease; and a traumatic influence, such as a fall, sometimes precedes the onset in such a way as to be significant. There is no proof of any relation between the onset of the disease and the process of dentition. Many children are perfectly well at the time at which they are attacked; few are in conspicuously feeble health. The disease has been thought to be occasionally secondary to acute febrile conditions, but the frequency of the relation has probably been overrated. The essential general disturbance associated with the disease itself has probably, in many cases, been mistaken for an independent acute febrile disorder.

Morbid anatomy.—The changes essentially consist in an acute inflammation of the grey substance of the anterior horns. Observations on the changes in the early stage are not very common. The changes in the anterior cornua in such a condition are widely spread, but slight in degree, and attain considerable intensity in one or more foci in the cervical or lumbar enlargement, or in both. At these spots the horn is softened, there may be hæmorrhagic infiltration, sometimes an actual cavity. There is extravasated blood massed along the vessels, and granule corpuscles and other products of degeneration are present. When the change is slighter there is an infiltration of leucocytes, but the nerve cells may be structurally intact, although swollen and granular. This appearance may extend from the horns into the adjacent lateral column, in which the nerve fibres nearest the grey matter may undergo degeneration. The posterior columns are always unaffected. Such changes indicate interstitial inflammation of the grey matter. But some observations have revealed alterations in the motor nerve cells only, the intervening substance being quite unaffected. This suggests that there may be two forms—an interstitial and a parenchymatous. At a later period—years it may be after the onset—the changes found correspond to those described. As a consequence of these, when the affection is unilateral, there is a marked difference in size between the two sides of the cord. The anterior nerve roots at the most affected part are small and grey, and degeneration may be traced along the nerve trunk. The muscular fibres have been found narrower than normal, and in a state of granular disintegration, with an increase of the nuclei of the sheath and of the interstitial tissue. In extreme cases the fibres may have completely disappeared, and their place be occupied by tracts of fibrous tissue. Here and there a normal fibre may be found, but in slighter cases some of the fibres may regain their normal appearance. As a rule, however, many fibres perish, and those which recover are smaller than normal. Muscular fibres are also seen that are larger than normal, and it may be that it indicates occasional true hypertrophy. The bones are not only shorter but also smaller, the compact substance more uniform, and the Haversian canals smaller. No observations have as yet been made in reference to the cases in which an acute polyneuritis is apparently associated with the spinal cord inflammation.

Pathology.—The disease is probably inflammatory in the great majority of instances, but the almost instantaneous onset in some suggests a vascular origin, probably hæmorrhage, in such cases. Signs of inflammation do not precede the onset, so that probably those cases cannot be placed in the pathological category of poliomyelitis. The lesion interrupts the motor path, and causes immediate palsy. The cells and the nerve plexuses of the grey matter are both damaged. Recovery of slightly damaged structures restores the voluntary path, either wholly or partially, so that the initial palsy is nearly always more extensive than the persistent atrophy. The occasional interference with the conducting paths in the white matter is the result of the extension of the inflammation, which may even spread through all the structures of the cord, so as to affect sensation as well as motion. But even in these cases there are never the acute trophic disturbances which would surely ensue in transverse myelitis. The true pathology of the malady is still obscure. The general symptoms make it almost certain that it is the result of a blood state. It is probable that the spinal lesion is not the cause of the constitutional disturbance, but that they are both the result of a common cause. We have no evidence of the nature of the blood state, nor have we any indication of the way in which the remarkable relation to season is determined, whether by predisposing the individual or favouring the development of some toxic agent. Of the predisposing influence of age there can be no doubt, and we may assume that in the conditions in which it occurs the structural development of the nervous system is complete, but the nerve elements lack the stability that comes only from continual use.

Symptoms.—The onset is usually acute, often with constitutional disturbance. Paralysis, at first widespread, afterwards passes away, except from a region in which the muscles rapidly atrophy, and in which more or less weakening and wasting persist. The general disturbance may be severe or slight. In most cases there is pyrexia, with headache, prostration, loss of appetite, and restlessness. Vomiting is not uncommon, and in some cases there are convulsions. The palsy develops, in most cases, in the course of this disturbance, but the constitutional symptoms occasionally succeed the onset. In some instances the initial symptoms are those of severe cerebral disturbance. In adults there is sometimes marked somnolence before the onset, and occasionally diplopia, giddiness, or even delirium. The cerebral symptoms pass away, together with the indications of general disturbance, and are probably the result of a blood state. In rare cases the onset is accompanied by symptoms of slight, transient, spinal meningitis.

In older children and adults, another common initial symptom is pain, sometimes referred to the muscles, sometimes to the limbs generally, occasionally occurring in the course of the nerves. It may be accompanied by extreme tenderness of the limbs, and depends upon the existence of a neuritis, which may come on at the same time as the palsy, although independently of it. Pain may also be felt in the back, dull and aching in character. The paralysis is almost always rapidly developed. It varies much in its initial range; only part of a limb may be affected, or there may be universal loss of power. But the extent of paralysis is usually intermediate between those extremes. When all four limbs are paralysed, the neck muscles also may be weak, and even swallowing may be impaired. Occasionally there is incontinence of urine, chiefly in severe cases; and although it is usually a transient symptom, it may last a long time. It occurs usually, of course, in cases in which the legs are affected from damage to the lumbar region. Paralysis may commence in one limb, and quickly spread; but very often the onset is so rapid, that all the parts are found to be paralysed when it is first discovered. Improvement is usually slow, and the permanent paralysis as a rule affects the limb which is first attacked.

Cases are sometimes seen in which the paralysis comes on suddenly, but without general disturbance. The suddenness of the onset makes it likely that the lesion is hæmorrhage, and not simple inflammation. But it is probable that even a primary extravasation of blood into the grey matter is due to the same causes that give rise to inflammation. In some cases of sudden onset, the initial palsy is universal, and in such it is not unlikely that a limited hæmorrhage in the cervical region causes rapid pressure on all the adjacent structures, and so determines the completeness of the paralysis. The structures, however, which have only suffered from the effects of the pressure, recover. And so the paralysis in those cases clears up to a great extent; and, indeed, the general rule is that the initial loss of power in cases of this disorder is far more extensive and severe than the permanent affection. In only a small number of cases do the two correspond. One general fact, however, is true of almost all cases, namely, that the palsy scarcely ever passes away entirely, and that where wasting occurs wasting remains.

When the atrophy is distinct, the muscles no longer contract to faradism, and the motor nerves are found to have lost their irritability. The change depends on degeneration of the nerves, and the muscles usually present degenerative reaction in its characteristic form. In the muscles which are weak, but do not waste, there is no degenerative reaction, although there may be slight diminution of excitability to each current. The paralysis, as a rule, is motor only; sensation is impaired in extremely rare cases, in which the inflammation is so intense as temporarily to interfere with all the conducting functions of the cord. Reflex action is necessarily lost in parts related to the affected muscle. No knee-jerk can be obtained, for example, if the extensors of the knee are affected even in slight degree. A common effect of the disease is to retard the growth of the bones, so that the limb gradually becomes shorter than its fellow; this effect is sometimes considerable, even when the atrophy is slight.

On account of the laxity of the tendons, corresponding to the paralysed muscles, the articular surfaces of the bones forming the joint are no longer in apposition, and repeated dislocation may easily occur. During the chronic stage of the disease there is a great tendency to the shortening of the muscles, and grave deformities may thus be produced. Posture also aids the production of these muscular contractions. Bedsores are almost unknown, even in the acute stage of the disease. The persistent paralysis may affect the whole or part of one or more limbs. The legs suffer three times as frequently as the arms, and there may be varying combinations of the parts of the limbs affected. The grouping of adjacent muscles in the affection is sometimes distinctly that of functional association. In the arm almost all the muscles are sometimes affected, but all are seldom entirely paralysed. The deltoid suffers more frequently than any other single muscle of the arm; occasionally the deltoid, supra- and infraspinatus, biceps, and supinators, are all involved in the upper-arm type of palsy of Erb. The irregularity of the grouping is shown by the fact that the triceps is often affected with these muscles. The neck muscles rarely suffer, but the diaphragm is occasionally paralysed. Paralysis of one side of the face, associated with wasting of the limbs, has been recorded.

The course of the disease comprises three stages—first, an initial one of paralysis; secondly, a stationary period; and, thirdly, a period during which the palsy passes away, except from the parts in which it is permanent. Even in the slightest cases, complete recovery is very rare. On the other hand, death from the disease is equally uncommon, and occurs chiefly in the early stage. Bronchial catarrh is an occasional cause of death when the respiratory muscles are affected. Relapses and sequelæ are both rare.

Diagnosis.—It is only in the early stage that difficulty can arise. As soon as wasting is distinct, the nature of the case is evident, and this is confirmed by the loss of reflex action, and the absence of sensory impairment. At the onset mistakes may readily be made. General disturbance is naturally ascribed to some general cause, and at first the existence of the paralysis and of the condition which determines it may be overlooked. But error will be avoided if it is remembered that mere prostration does not cause total or local inability. In adults the danger of mistakes is less, but the rheumatoid pains, which are so common, may be misinterpreted. Of other spinal cord diseases, all chronic lesions are distinguished by the mode of onset. Acute transverse myelitis is simulated when the disease of the grey matter is bilateral and so intense as to extend into the white columns. A real difficulty is confined to adults, in whom there is extensive cervical or lumbar myelitis involving the grey matter. In young adults, poliomyelitis is more likely. The distinction of diphtheritic paralysis and of multiple neuritis is from the subacute rather than the acute spinal atrophies. Diagnosis of paralysis from cerebral origin is usually easy. In the latter there is no loss of faradic irritability, nor is there extreme wasting or loss of reflex action. Any cerebral symptoms which may attend the onset of poliomyelitis are subordinate in significance to the state of the muscles. Convulsions at the onset of infantile spinal paralysis are general; those resulting from a cerebral condition are usually unilateral or commence locally. The exceedingly slow onset of pseudo-hypertrophic paralysis ought to render its confusion with poliomyelitis impossible. Diseases outside the nervous system, which have been mistaken for infantile paralysis, are chiefly those in which pain interferes with the movement of the limbs, such as hip-joint disease, necrosis of the femur, scorbutic rickets; but in all such cases examination will reveal the absence of actual paralysis, and that there is no interference with reflex action.

Prognosis.—Danger to life exists at the onset, and is usually in proportion to the amount of constitutional disturbance. In the stage of paralysis, interference with respiration constitutes the only danger. But in the great majority of cases the disease may be looked upon as involving little or no risk to life. As soon as the paralysis has become stationary, the question arises, and is anxiously asked, what will be the permanent condition? An answer cannot be given until the end of the first week or ten days, and then only by means of an electric examination. Whatever muscles have lost faradic irritability at the end of that time will certainly waste, will remain for a long time paralysed, and will probably be permanently affected in some degree. On the other hand, if there is no loss of irritability at the end of ten days, the wasting will certainly be slighter, and some ultimate recovery may be expected, even in the most affected part. When there is no loss of irritability, the paralysis will probably pass away in the course of a few weeks. The return of faradic irritability that has been lost is a favourable indication. In the chronic stage the prospect of ultimate recovery depends on the rate at which wasting developed, on the electrical reaction, and the duration of the case. Where there is no sign of returning power at the end of three months, very little recovery will occur. On the other hand, if at the end of one or two months some faradic irritability can still be detected, improvement is probable, and may be considerable. It is necessary, in the case of children, to remember the likelihood of the bones suffering, and that shortening of the limb is therefore not improbable.

Treatment.—In the initial stage, treatment should be that of the general state. Free sweating, and the use of salicylate of soda or salicin will be indicated in a case distinctly due to cold. At present we have no means of counteracting the morbid blood state which is probably present. The child should be kept at perfect rest on the side, and warmth applied over the affected part of the cord by poultices or fomentations. In such a disease there is great difficulty in ascertaining the effect of treatment on a lesion of the cord. Full doses of ergot and belladonna have been credited with a control or arrest of the morbid process. But the good result of such treatment is probably a mere coincidence. After the acute onset is over, and the spinal lesion becomes stationary, great care is needed in watching the constitutional condition. Perfect rest should be maintained for some weeks; this more especially when there is persistent tenderness, the result of an independent neuritis. When the acute stage is over, strychnine may be given in all cases, but it should be commenced only two to four weeks after the disease has become stationary. It is probably unnecessary and undesirable to give it by hypodermic injection in this disease. If the child is rachitic, the drugs that have most influence in counteracting the condition of rickets, especially cod-liver oil, iron, and the lactophosphate of lime, should be administered. The use of electricity is very important in this disease. While the influence normally exerted through the nerve fibres is in abeyance, the muscles are without the function of stimulation, without the exercise which they need. In such a condition, the use of electricity to cause contraction, and so produce functional stimulation, is clearly indicated. Wherever cell and fibre have perished, nothing that electricity effects can be of service. When there has been damage but not destruction, stimulation of the muscles by the voltaic current will keep them in a favourable state of nutrition, ready to resume their function as soon as the nerves are capable of transmitting impulses. The mode of application should be by repeatedly stroking the muscles with one terminal (a sponge or conductor, covered with water-holding material, well wetted with salt and water), which should be lifted from the skin between each stroke. The other terminal is kept still, and is best placed on the upper part of the muscle where its nerve enters it. The emotional disturbance interfering with the treatment can generally be avoided by commencing with a very weak current. It is better, indeed, to commence with no current at all, applying the sponges in the way directed, so that the child may be familiar with them and cease to fear them. If, in spite of these precautions, the strength necessary to obtain muscular contraction cannot be employed without distressing the child, the attempt should not be persevered in, and only such a current used as does not disturb the patient. Electrical treatment may be commenced at the end of the third or fourth week. It is unwise to use it earlier, because of the danger there may be of exciting increased disturbance in the cord. Another measure which should be employed is systematic upward rubbing and gentle kneading of the limbs, and great care should be taken at all times to keep them warm. When some voluntary power is regained, systematic planned movements to exercise the muscles are of great importance. Such movements have to be specially arranged to meet the exigencies of individual cases. A very important element in the management of the chronic stage is the prevention and treatment of the muscular contractions and deformity. The contractions cannot be entirely hindered, but may be often prevented from reaching a high degree by careful attention to position. For developed deformities, due to great shortening, surgical treatment by splints or tenotomy is usually necessary. For this and other reasons, mechanical appliances are of undoubted value; by counteracting deformities and supplementing weak muscles they may often enable a child to walk, but in all these cases the instruments require frequent attention and alteration to adapt them to the growth of the child, and patience is necessary in the management of those cases through their long and tedious course. At the end of a year the lesion of the cord has become a cicatrix, and further improvement will be merely by the slow growth of the muscle that has recovered under the stimulus of use. And this improvement will go on, whatever treatment is adopted, and whether special measures are employed or not, so long as care is taken that deformity and contraction are not allowed to interfere with the proper use and exercise of the affected parts.

PROGRESSIVE MUSCULAR ATROPHY.

Syn., Wasting Palsy; Chronic Poliomyelitis; Amyotrophic Lateral Sclerosis.

This disease is characterised by slow wasting of the muscles, usually spreading and increasing until it is wide in extent and extreme in degree. The changes in the muscles are correlated with changes in the spinal cord, and degeneration in the motor nerve fibres. Lockhart Clarke was the first to discover disease of the grey substance of the cord; Charcot especially associated the atrophy with the wasting of the ganglion cells. Two varieties of the disease have been distinguished by Charcot. In the one, there is wasting of the muscles; in the other, in addition to this, indications of degeneration of the pyramidal tracts of the cord. In the latter class Charcot regarded degeneration of the pyramidal tracts as primary, and the affection of the horns as secondary. He therefore termed cases of this class deuteropathic, and applied to them the name amyotrophic lateral sclerosis, and cases of the first class he called protopathic. But doubts have been expressed as to the validity of the distinction, and it is likely that the two types are to be regarded as extreme examples at the opposite ends of a series, and cases are met with that present every gradation between the two.

Etiology.—The disease is chiefly one of adult life, and it affects males more frequently than females. Heredity is not to be traced in more than half the cases, and generally as an indirect neuropathic disposition. Rarely there is direct inheritance of the disease. Of the more direct causes the most frequent is probably mental distress and anxiety, and this is met with especially in females in late life. Another cause is exposure to wet cold, either habitual or accidental, and the onset after such exposure is usually ushered in by pain. Injury to the cord is a rare cause. Still more rarely does a fall injuring one limb produce it. But in such a case the muscular atrophy commences in the limb which has been injured. The disease sometimes succeeds syphilis, and no other cause may be traceable; but it is noteworthy that in at least some of the cases the wasting commences during or directly after energetic mercurial treatment. In the cases in which atrophy is followed by acute specific disease, such as measles, it is not unlikely that the malady is a chronic neuritis. General muscular atrophy may result from lead-poisoning, but in many cases of progressive muscular atrophy no cause of the disease can be traced.

Morbid anatomy.—The muscles are reduced in size, and pale in colour. Sometimes there is little in the tint of what remains to suggest muscular tissue; but, on the other hand, the mass of the muscle may be dark, and pale streaks in it may indicate the arrest of degeneration. Under the microscope there may be—(1) simple narrowing of fibres, without any considerable change in the striation; (2) simple fatty degeneration, the transverse striation being replaced by a granular appearance; (3) the fibre sheath may contain only clear material, enclosing a few fatty globules and a few transverse striæ, probably the result of the dissolution or vitreous degeneration of the striæ; (4) the longitudinal striation at first coexisting with the transverse, but ultimately occupying the fibre alone. Occasionally, however, along with the longitudinal striation, there is transverse striation, very much finer than normal; but this change may be present in only one part of the fasciculus. The peripheral nerves contain many degenerated nerve fibres, and the terminal branches for the muscles a much larger number. These degenerated nerve fibres are found to come only from the anterior roots. The posterior roots are normal.

The spinal cord is often softer than natural at the affected part. Under the microscope, morbid changes are seen in the anterior cornua, and also, in most cases, probably in all, in the anterior and lateral white columns. The change in the anterior horns corresponds in its intensity to the degree of affection in the corresponding peripheral tissues. Most of the large nerve cells have disappeared, either entirely or so as to be represented only by small angular bodies. The interstitial tissue is also changed; the nerve fibrillæ waste with the cells, and there is an increase of the small angular and stellate cells and other connective tissue elements. The larger vessels are dilated, and so are the capillaries. The cornu, as a whole, is not changed, either in size or shape. Similar alterations may be traced through the dorsal region. In the lumbar enlargement the grey matter may be normal if the legs are unaffected, or altered in a manner similar to that described above, if the atrophy has affected the lower limbs. And if the atrophy begins in the legs, the change in the lumbar cord may be correspondingly greater. There is degeneration of the anterior root fibres, and also of the fibres of the anterior commissure. In the white columns there is usually considerable, often almost complete, degeneration of the pyramidal tracts, anterior and lateral. Where the direct cerebellar tract exists, this always limits the sclerosis in the lateral tract on the outer side. On the inner side, the lateral limiting layer is usually much less degenerated than the pyramidal tract, and separates this from the grey matter; but this layer itself suffers in some degree. The sclerosis also usually extends forward in the mixed zone of the lateral column, lessening in intensity, and usually ceasing opposite the anterior part of the anterior cornu. Occasionally it extends into the anterior columns. The posterior columns are always free from definite degeneration, but there may be some general increase of connective tissue throughout the cord. When the sclerosis is intense, it can be traced up through the decussation to the medulla oblongata, in which both pyramids may be degenerated; and this degeneration may even extend through the pons and cross to the internal capsule, and even through the white substance, to affect the cortex. Here the large ganglion cells have been found fewer than normal, and distinctly degenerated. In cases in which the symptoms of bulbar palsy were present during life, the motor nuclei of the medulla have presented changes similar to those in the grey matter of the spinal cord. In other instances the affection of these nuclei has been slight, but in such cases the degeneration of the pyramids has been intense, and doubtless involved the fibres connecting the bulbar nuclei with the cortex.

Pathology.—The constancy of the changes in the ganglion cells of the spinal cord, and the degeneration of the motor root fibres, leave no doubt of the relation of the muscular wasting to disease of the grey matter. The slowness of the change causes the impairment of the muscular fibres and of the nerve elements to proceed *pari passu*, and we are thus able to understand the slow failure in electrical excitability, as nerve and muscle fail together. The essential lesion of the disease is probably an affection of the whole motor path, manifesting itself first in what is known as the lower motor segment, consisting of ganglion cells and nerve fibres. The muscular wasting is secondary. As has been pointed out, the upper segment is often degenerated as well as the lower, a degeneration which is most distinctly manifested in the pyramidal fibres. The degeneration of the upper and lower segments is simultaneous, and are both the results of the same tendency to degeneration of the motor path. Such a view of the pathology explains the varying clinical conditions met with, and especially the irregular combination of tonic and atonic atrophy, with signs of increased reflex excitability, which have been already referred to. The cases in which the legs present the simple palsy and spasm of the tonic atrophy have been separated by Charcot, as has been already said, into a group, which he has named amyotrophic lateral sclerosis, the separation being based on the assumption that in such cases the primary lesion is a degeneration of the pyramidal tract, and the affection of the grey matter secondary. But, as we have already stated, the cases of this disease constitute, in reality, one prolonged series, with all variations between muscular atrophy with flaccidity, and diminution or loss of reflexes, and atrophy as severe, but combined with rigidity and increased reflex excitability, such clinical difference being correlated with the slight or severe involvement of the upper motor segment. The process in the grey matter has been regarded by some as a chronic inflammation, but it is to be more correctly regarded as a degenerative disease. But of course, whatever be the nature of the primary process, we must recognise secondary tissue changes of independent energy, and be prepared to admit that the distinct presence of inflammation may occasionally form part of these.

Symptoms.—The definite symptoms of the disease are often preceded by aching pains in a part afterwards wasted; sometimes, also, there is pain in the spine, and there may be a sense of coldness preceding the atrophy in a particular limb. Weakness and wasting usually come on together. In the shoulder and back the loss is usually first noticed, while the wasting is often observed first in the hand. The affected muscles lose their proper shape; there is flattening, or even a depression where there should be a prominence.

The disease commences in the arm in nine-tenths of the cases, as frequently in one arm as in the other, and with almost equal frequency in the hand and shoulder muscles. As the muscles waste, paralysis of varying character and degree results. In the hand, the thenar muscles and the interossei are usually the first to suffer. Of the interossei, the abductor indicis is conspicuously affected. The forearm muscles may be next involved, the flexors usually before the extensors. The supinators may suffer at the same time, or they may escape until the biceps is involved. Occasionally the disease begins in the forearm, and then especially in the extensor muscles of the fingers. Of the shoulder muscles, the deltoid usually first manifests the disease. It is not rare for one part of the deltoid to escape while the other suffers. The triceps usually suffers less and later than the biceps, but occasionally the reverse is the case. In exceptional instances the deltoid or hand muscle has escaped, and there are cases in which the upper arm and shoulder are affected, while the hand and the forearm escape.

In most cases, the muscles of the back are early involved in wasting. The middle and lower parts of the trapezius usually suffer first, the rhomboids and the erectors of the spine later. The highest part of the trapezius shows a remarkable resistance to atrophy, and was termed on this account by Duchenne the *ultimum moriens.* Yet, in exceptional cases, the highest part of the trapezius suffers more than the middle. The muscles supporting the head frequently suffer, and lead to difficulty in carrying it; and even in bed the patient may be unable to move his head from side to side. The muscles of respiration suffer in the great majority of cases, the intercostals, as a rule, more than the diaphragm, although the diaphragm may be affected, while the intercostals escape. The abdominal muscles are not so liable to be affected as those of the thorax, and wasting in the legs is much less common than in the arms. The face nearly always escapes the general atrophy, and its normal appearance may be in striking contrast to the rest of the body. In many cases, however, the lips, tongue, and palate are paralysed, as part of the bulbar paralysis which so often forms an integral part of the disease. As the wasting progresses, the appearance of the parts undergoes great changes. The electrical irritability of the muscles varies in different cases. When the wasting is slow, there is usually diminution in both faradic and voltaic irritability, similar in character to each current. When the wasting is extreme, the irritability at last becomes extinct; but the voltaic irritability persists long after the faradic has disappeared. It may be normal in degree, or lowered, but is seldom increased in such cases, and there may be no qualitative change. The mechanical irritability of the muscles is considerably increased, and spontaneous flickering contractions are very common, but not invariable.

The reflex action is different in two different classes of cases. In the class of cases at one extremity of the series, the knee-jerk is excessive, and ankle clonus is present, even where the muscles are in some degree wasted. In the class of cases at the other end of the series, the knee-jerk is normal, or may be lost, even although the wasting of muscles is only slight. In this class the muscles are usually flaccid and toneless, while in the other class the muscles are rigid and firm. The conditions have been named tonic atrophy on the one hand, and atonic atrophy on the other. These varying conditions will be referred to when we consider the pathology of this condition. Sensory symptoms are usually slight and unimportant. Cutaneous sensibility is never impaired, nor is there any impairment of the muscular sense. The combinations of the ordinary atonic wasting, and loss of power without wasting, and with increased myotatic irritability, are very numerous and variable. The most frequent combination is that of flaccid atrophy in the arms, and spastic paralysis in the legs. But there may even be atonic atrophy below the knees, with well-nourished thigh muscles and a much-increased knee-jerk. The extreme emaciation of the most affected parts shows that the adipose tissue wastes as well as the muscles. The atrophied muscles are usually cold, but there is, as a rule, no tendency to acute vasomotor disturbance. In exceptional cases certain areas of muscles may undergo wasting, the rest being normal. The functions of the sympathetic are not, as a rule, deranged. Dilatation and contraction of one pupil have been frequently observed in association with atrophy of the muscles that are supplied from the lower part of the cervical region. This, no doubt, depends on disease of the spinal cord, not of the sympathetic itself. The reflex action of the pupil is usually normal, and optic nerve atrophy never occurs. Nystagmus is present in rare cases. The visceral functions may be little disturbed, but sexual power is often lost. The sphincters are unaffected.

Complications.—Progressive muscular atrophy is occasionally accompanied by the symptoms of some other degenerative disease of the cord. In rare cases, atrophy in the arms is accompanied by signs of locomotor ataxy in the legs; but by far the most frequent complication is bulbar paralysis, although perhaps it is scarcely correct to speak of it as a complication, but rather as the expression of the same degenerative tendency in another, but analogous, part of the nervous system, the bulbar nuclei being the analogues at a higher level of the anterior horn cells of the spinal cord. The bulbar palsy occurring in this disease is practically identical in characters with that which occurs in the isolated form. Frequently, however, there is but little paralysis of the tongue, even when swallowing and articulation are much impaired. There may also be slight interference with articulation when there is no marked bulbar palsy. General paralysis of the insane has been met with as an exceptional complication.

The malady is usually progressive. It may, however, become stationary at any period in its course, and, when once arrested, it does not usually again become active. The tendency to cessation is greatest in a later stage of the disease, when there is little except life to be preserved. Sometimes progress ceases at an earlier stage, chiefly perhaps in those cases in which the atrophy is strictly symmetrical. Occasionally some symptom, such as weakness of the sphincters, may disappear, although the atrophy progresses. When the progress at the commencement is rapid, it usually continues rapid; when it begins slowly, it is usually slow throughout. An interval of a year may intervene between the affection of the two arms; in one case the interval has been as long as seven years. Probably the shortest time in which a patient has reached the last stage of the disease is about nine months. The uniform progress of the disease may be interrupted by the almost sudden palsy in a certain group of muscles. The extensors of the wrists and fingers are those most commonly affected thus, and the weakness is usually followed by a distinct degenerative reaction in the muscles. When there is weakness in the legs, without wasting, the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. The chief danger to life is from pulmonary maladies, and the complication mentioned of bulbar paralysis increases the risk of such an affection. Less commonly, death results from bedsores, or intercurrent maladies.

Diagnosis.—The diagnosis of the developed malady is simple. At the onset, when the muscular wasting is limited, it is sometimes not easy to decide whether the atrophy is local, or the expression of a general tendency. The wasting from disease of a single nerve or collection of nerves in a plexus is sufficiently distinguished by its limitation. Much more difficult is the distinction from some forms of multiple neuritis. It is often necessary to wait before coming to a definite decision; but in not a few cases, careful search will reveal other symptoms of neuritis, and a known cause is usually obtrusive. In pachymeningitis of the cervical region, the wasting is less chronic in onset, and is always accompanied by distinctive sensory signs, acute pains, and usually irregular anæsthesia. The same distinctions mark diseases of the nerve roots of the cauda equina.

Chronic disseminated myelitis may cause widespread muscular atrophy, but it is distinguished by symptoms of irregular damage to other structures. The diagnosis from syringomyelia is to be made by recognising the sensory and trophic changes which occur in the latter disease. From idiopathic muscular atrophy, the distinction is sometimes easy, occasionally very difficult. It is chiefly difficult in the atrophic cases without enlargement, particularly in those which commence in adult life. It will be better understood after perusal of the account of that disease.

Prognosis.—The chief guide is the observed tendency of the morbid process. It is usually progressive; but there is a possibility of arrest, which is greater in middle life. The prospect of arrest seems to be greater in the cases in which the wasting is symmetrical. Spontaneous cessation occasionally occurs, but seldom before an advanced stage of the disease is reached. Arrest may take place, as a result of treatment, at any stage. After arrest, a certain degree of improvement may result; but wasting that has existed for six months will probably persist unchanged.

Treatment.—The general health must be carefully looked after. Fresh air, gentle exercise without fatigue, and the absence of all mental strain, are most important. If the patient becomes helpless, great care is necessary. Bedsores in this disease result from bad nursing, and are therefore avoidable. Undoubtedly the most efficacious treatment is the administration of strychnine by hypodermic injection. The evidence of arrest consequent upon this treatment is now undoubted, and it should be remembered that the hypodermic administration of the drug has been successful in cases in which it has failed to affect the disease when given by the mouth. It is conceivable that the different result is due to the fact that the agent is brought into more rapid contact with the nerve elements. One injection daily should be given, and the nitrate is the most convenient salt. It should be given at first in a dose of $\frac{1}{100}$ gr., quickly increased to $\frac{1}{40}$, or more. When the malady is apparently arrested, it is wise to omit the injections for one week in three or four. Other nervine tonics may be given by the mouth at the same time, for in a malady so grave it is desirable to neglect nothing that may exert a good influence. Local treatment of the muscles has but little influence on the wasting, the most sedulous and skilful use of electricity often fails to produce any effect upon the course of the disease. It is possible, however, that it sometimes does a little good. Patients find it hard to believe that this remedy cannot help them, and it may be well to give it a careful trial. As a general rule, voltaism is to be recommended; but in cases in which the muscles react to faradism it is occasionally advisable to use that current. The application should always be one of moderate strength, for strong applications often cause much subsequent pain. Rubbing and massage of the muscles often have an influence which can be said to be in the right direction, although its effect on the muscular atrophy is not, as a rule, very obvious. Combined with passive movement, however, its influence in preventing and diminishing deformities is most distinct. When the disease occurs in subjects of syphilis, specific treatment invariably fails, and cases are on record in which the progress of the disease seems to have been distinctly accelerated by an energetic course of antisyphilitic remedies.

PRIMARY SPASTIC PARAPLEGIA.

Syn., Primary Lateral Sclerosis.

Etiology.—An inherited neuropathic tendency is to be occasionally traced, and not uncommonly such a tendency manifests itself in mental disease. The disease affects both sexes, usually begins between 20 and 40, but may commence as late as 61. It sometimes follows syphilis in such a way as to suggest a causal connection. But the most frequent apparent cause is concussion of the spine, such as by a fall on the back. Some time may elapse between the fall and the first pronounced symptoms of the disease, and hence focal lesions due to the fall can be excluded. Next in frequency is repeated exposure to wet cold. Rarely the symptoms have followed some acute illness, and in several cases the disease has succeeded prostration after childbirth or abortion, or has commenced during lactation. The cause of the congenital form is always injury to the brain during birth, in most cases meningeal hæmorrhage.

Morbid anatomy.—Morbid anatomy is silent on the subject of uncomplicated lateral sclerosis. Complete degeneration of the pyramidal tracts, anterior as well as lateral, is met with chiefly in cases in which the anterior ganglion cells and motor nerves are diseased, although in some instances the amount of this disease is small and limited. In the lumbar region such degeneration is usually limited to the lateral columns. The anterior tract may be affected higher up the cord. The lateral sclerosis is bounded externally by the normal direct cerebellar tract, and internally by the lateral limiting layer; and there is the usual increase of connective tissue and wasting of nerve fibres. The degeneration probably begins, in all cases, in the nerve elements themselves, and it has been traced, through the medulla, pons, and hemispheres, to the motor cortex, in which indications of degeneration have also been found. The direct cerebellar tract and posterior vesicular columns have also been found degenerated, and an annular sclerosis of the periphery of the antero-lateral columns has also been observed. More frequent, however, is a combination with degeneration of the posterior columns, described under Ataxic Paraplegia. Another point that deserves mention is the occasional occurrence of lateral sclerosis in association with insular sclerosis. A similar association of insular and posterior sclerosis has also been observed. It seems probable that lateral sclerosis, although a system disease, is often one of less strict features than tabes, and has some tendency towards chronic myelitis. The mechanism of the symptoms will be obvious from a consideration of the changes on the spinal cord.

Pathology.—The term primary lateral sclerosis indicates the supposed nature of the change in the spinal cord underlying the symptoms of this disease. Although absolute demonstration has not yet been furnished, the indirect evidence of the correctness of the pathological hypothesis is very strong, and the only open question is whether, when there is no disease of the motor nerve cells of the anterior horn, the degeneration is so limited to the pyramidal tracts as to constitute a system disease, in the strict sense of the word. It should, however, be noted that cases which present this group of symptoms may be very various in nature. When the upper motor segment—that is to say, the part comprised between the cortex and the anterior horn cells of the cord—

is involved, the symptoms are the same in whatever part of the segment the disease is situated. Thus, if there is disease of the leg centres in both hemispheres, the state of the legs may be identical with that resulting from disease of the spinal cord. Such bilateral disease often results from injury during birth—meningeal hæmorrhage over the upper parts of the central convolutions. This condition is termed congenital spastic paraplegia. On the other hand, degeneration may begin in the termination of the fibres in the grey matter, and may even be limited to these terminal parts. In such a case the white columns will be found normal, as in one recorded case, in which the symptoms of spastic paraplegia existed during life, and no anatomical change was discovered after death. It is highly probable that the toxic agents which cause lathyrism and pellagra act on the extremities of the pyramidal fibres, as curare does on the nerves. The clinical and pathological evidence shows that the morbid state often occurs as part of a more extensive degeneration.

Symptoms.—The first symptom is weakness of the legs, of gradual development. Progress of the weakness is variable, but in all characteristic cases it is slow. After even several years the patient is still able to walk a mile or two, slowly no doubt, and with difficulty. But, on the other hand, walking power may be almost lost at the end of six months. The early weakness is not uncommonly accompanied by some unsteadiness, but this is chiefly subjective. When the patient seeks advice, it will generally be found that there is distinct loss of power in the flexors, especially of the hip, but often much greater of the ankle than elsewhere. The knee-jerk is quick and excessive; foot clonus is usually obtained with readiness. The rigidity is noticed as slight stiffness of the legs on first rising; this gradually increases as power lessens, until the spasm may be such that the knee cannot be passively flexed by any force that can be applied to it until the spasm becomes less. When flexed, the limb is comparatively supple; but if it is then extended, the spasm instantly returns, and often completes the extension, just as the blade of a knife opens out under the influence of the spring. And thus is produced the so-called clasp-knife rigidity. Occasionally there are paroxysms of brief flexor spasm, especially during sleep. When this is great, it may pass into clonic spasm. Reflex action from the skin is also usually excessive, and the attempt to evoke it may excite an attack of spasm. The gait is very characteristic, the legs seeming to drag behind the patient, and each is hauled forward as a rigid whole, the toes catching against the ground, and the limb may shake from the clonus developed by the extension of the calf muscles. The muscles themselves are usually large and well nourished, and often, indeed, seem to be hypertrophied. It may be that sometimes they really are so; but they are not always large, and are indeed sometimes moderately wasted. Electrical irritability is usually perfectly normal. After the patient has ceased to walk, contracture sometimes occurs in the calf muscles. Similarly, flexor contraction of hip or knee may occur under the influence of posture.

The arms are often unaffected, but they suffer in most cases from similar progressive weakness and excessive myotatic irritability. A clonus can sometimes be obtained in the flexors of the fingers; there is less paroxysmal spasm than in the leg, much rigidity of the limb, similar to the late rigidity of hemiplegia. The difference in the form of spasm in the arm and leg is no doubt connected with the existence of a reflex extensor mechanism in the lumbar centres concerned in the act of standing. The muscles of the arms are often as well nourished as those of the legs. The affection of the two arms, as of the two legs, is rarely equal. Sometimes, however, the arm and leg on one side are paralysed, while the limbs on the other side are comparatively slightly affected. The muscles of the trunk may also suffer, and spasm of the abdominal muscles may occur in severe cases. It is not common, in typical cases, for the symptoms to extend into the region of the cranial nerves; but in rare instances difficulty of swallowing and of articulation has existed, and excessive myotatic irritability has been observed in the muscles of mastication, so that a tap on the chin causes a vigorous elevation of the depressed jaw, or may even produce a jaw clonus. Sensory symptoms, with the exception of slight dull pains in the legs or back, are generally absent. Rheumatoid pains may be troublesome, sharp pain is rare. Subjective sensations of numbness, tingling, and formication are more common, and a girdle sensation may be present. The sphincters may be affected, and occasionally suffer very early. Sexual power may be lost, or may remain even when the paraplegia has reached a high degree. Ocular symptoms are rare, with the exception of nystagmus, which will often be found if looked for. The action of the pupils is usually perfect, and, in uncomplicated cases, optic atrophy is extremely rare. Transitional forms are met with, which constitute connecting links between the primary spastic paraplegia and other forms of paraplegia. Slight inco-ordination may exist in cases thus approximating to the combined postero-lateral sclerosis. Or slight muscular wasting in the arms may be associated with indications of lateral sclerosis in the legs, constituting a connecting link between primary lateral sclerosis and amyotrophic lateral sclerosis. In some cases the disease is the expression of the general morbid tendency which underlies general paralysis of the insane; and in many such cases the mental symptoms may be extremely slight. The course of characteristic cases is a very chronic one, and it is perhaps the least dangerous to life of any chronic spinal disease. Even secondary kidney trouble scarcely ever occurs, perhaps because the excessive reflex action may save the bladder from injurious over-distension.

Diagnosis.—The diagnosis rests on the combination of the weakness, excessive reflexes, spasm of gradual onset, and also on the absence of indications of a focal lesion. Spastic paraplegia is common after such lesions, but is secondary, not primary. The absence of unsteadiness is the chief distinction from ataxic paraplegia. Considerable muscular wasting in any part is probably to be regarded as bringing the case into the category of amyotrophic lateral sclerosis. Primary spastic paralysis is not uncommon at the age and in the sex in which hysteria prevails, and there is no form of cord disease which is so often mistaken for hysterical paraplegia, owing to disregard of the evidence afforded by the foot clonus, but the opposite error from undue regard to this symptom is practically unknown. When the arm and leg suffer on one side only, the disease may be mistaken for cerebral hemiplegia. There is not, however, any affection of the face, and the limbs on the other side are never quite normal. The diagnosis of the congenital infantile form is only difficult when the observer is unaware of the occurrence of these cases. A slight degree of inco-ordination in the hands will usually be found if they are carefully watched. Chronic primary cord diseases are almost unknown in young children. Caries of the spine is, in them, the chief cause of paraplegia, and the definite onset of the paralysis in a previously healthy child is an absolute distinction from a case of birth palsy.

Prognosis.—Actual recovery is rare, but does sometimes occur. In cases that improve, it is possible that the disease is limited to the terminal structures. We have not at present any guide to the prognosis in an individual case, except its duration and degree, and its observed tendency.

Treatment.—The influence of drugs is not always appreciable in this disease. Nux vomica and strychnine have to be given with caution, as they rather tend to increase the spasm. In severe cases this is a most distressing symptom, often not amenable to any influence. Bromides sometimes lessen it. Indian hemp, belladonna, and Calabar bean have seldom a very distinct influence. Absolute rest is sometimes of service, and the avoidance of fatiguing exertion is a very important element in the treatment. Upward rubbing seems to have more influence than kneading the muscles. If there is contracture of the calf muscles, the foot should be pressed up while these are rubbed. In one case of moderate degree, almost all the symptoms passed away after a course of Turkish baths, arsenic being administered internally. Electricity is useless in the pure disease, and may do harm by stimulating the already excessive excitability of reflex centres. In the infantile form, drugs are useless, rubbing is desirable, and passive movements calculated to minimise or prevent deformities.

LOCOMOTOR ATAXY.

The name locomotor ataxy was given by Duchenne to a chronic malady in which there is inco-ordination of muscular contractions, without loss of power, manifested more often in the legs than in the arms, and thus rendering walking first unsteady, and afterwards almost impossible. The disease had previously been distinguished by Todd, who also discovered its relation to degeneration of the posterior columns of the spinal cord. But it has been found that other distinctive symptoms may precede inco-ordination, and hence the term tabes dorsalis, an old name for paraplegia, sufficiently unmeaning to be devoid of erroneous significance, has become generally used. Until lately the disease was thought to be an affection of the spinal cord alone, but peripheral degeneration of the sensory nerves has been found. The posterior roots are also degenerated, and the median part of the posterior column, which is specially affected, contains fibres which pass up from the roots without interruption. Hence the disease must be regarded as essentially a degeneration of the neurons related to the cells of the posterior spinal ganglia, each of which consists of an efferent and an afferent fibre, with the cell of which they are the processes, a unipolar cell, because the two processes are, for a short distance, conjoined.

Etiology.—Such isolated degenerations are generally the result of some special poison circulating in the blood, perhaps for a long time, and acting on the structures particularly susceptible to it. The majority of the cases of tabes seem to be due to a toxine produced by syphilis. The evidence is that they are preceded by syphilis, that they cannot be cured by the treatment which arrests and removes the true syphilitic processes, and that the same condition may be caused by other toxic agents, by that which is associated with diphtheria, and also by alcohol and arsenic. We know the nature of the first, that it is a chemical poison, only indirectly due to the organisms of the specific disease, and we are therefore justified in concluding that a similar agency is at work in the more frequent cases in which tabes follows the more chronic specific disease, syphilis. This can be traced as an antecedent in more than two-thirds of the cases, and it is notorious that syphilis often occurs when it is not traceable. It must be regarded as the remote cause of at least four-fifths of the cases. The effects of the other poisons just mentioned pass away, and the conditions which they produce are therefore termed "pseudo-tabes." Of the remaining fifth, or less, of true tabes, some are probably due to other toxines, of which we have, as yet, scarcely any glimpse. Some are possibly due to senile degeneration. There is much reason to doubt the influence of falls and blows, which have been alleged as causes; and exposure to cold, if effective, is doubtless so through a toxic mechanism.

Morbid anatomy.—In most cases the spinal cord presents changes visible to the naked eye, the posterior columns having a grey translucent appearance. It may affect the whole of the columns, or only parts of them. The posterior columns are also smaller than normal, hence the shape of the cord is slightly changed. In a section it is seen that there is an excess of connective tissue in the affected areas. In the most common condition, in which the legs only are affected, this sclerosis occupies the whole of the columns in the lumbar region, but it is often slight in the anterior parts of the postero-external columns. Above the lumbar enlargement the affection of the postero-external columns gradually ceases. In the cervical enlargement, the unaffected area in the posterior part of the postero-external columns is usually much larger than in the lumbar enlargement. In rare cases, of long duration, the posterior columns are replaced by connective tissue in the whole extent of the cord. In slight cases, on the other hand, the sclerosis is moderate in degree, even in the regions of the cord most affected. The tract of fine fibres of the posterior root discovered by Lissauer is generally diseased. Sometimes there is distinct sclerosis of other definite tracts, such as the antero-lateral tract, or the direct cerebellar tract, and, rarely, there is distinct degeneration of the pyramidal tracts. The whole periphery of the antero-lateral column is thus occasionally the seat of sclerosis, the connective tissue extending in from the pia mater, which is usually also thickened. It is difficult to detect any changes in the grey matter, although it is likely that there is some atrophy of the nerve cells and fibres in the posterior horn in many cases. In more advanced cases various structural changes can be discerned. The posterior commissure may be reduced in size, the posterior vesicular column may show distinct degeneration, and some atrophy of the plexus of fine fibres in this column is said to be almost invariable. Frequently the cells and vertical fibres waste, and this atrophy of the cells is said to be specially associated with sclerosis of the direct cerebellar tract. When the cells are unaffected, the cerebellar tract may be normal. The degeneration of the grey matter often extends into the intermediate region between the cornua. The intermediate lateral tract of cells may atrophy, and even the anterior cornu and its cells suffer in a similar way.

The sclerosis varies considerably in its degree. The larger tracts are more fibrous than normal, more nuclei are seen than in health, but everywhere the new growth of connective tissue proceeds from pre-existing tracts. From the parts most changed the nerve fibres have entirely disappeared. In less affected tracts, close examination reveals many fibres narrower than normal, and stained points that may be axis cylinders. The vessel walls are usually thickened, chiefly as regards the outer coat and adventitia, and the pia mater is sometimes thickened over the posterior columns, and the vessels in it may be similarly changed. This peripheral sclerosis probably results in three ways—(*a*) primary degeneration of peripheral ascending tracts, (*b*) a process of chronic interstitial myelitis, spreading from the pia mater, causing, (*c*) a secondary degeneration of the fibres affected in the first process. The posterior nerve roots may appear normal in slight cases; often their disease is conspicuous. When the cord is much affected, they are invariably involved. They may be atrophied, thin, and grey to the naked eye, while under the microscope the nerve fibres are seen to be wasted, and the connective tissue to be increased. The changes extend to the ganglia, but cease there. The anterior nerve roots are normal, except in the rare cases in which the anterior horns have suffered.

The peripheral spinal nerves have been found degenerated, their fibres being wasted, and ultimately the axis cylinders perish. There may be a slight increase in the interstitial tissue and nuclei; the change commences in the nerve fibres themselves. The degeneration is greatest in the terminal nerve filaments, and the sensory fibres seem to be exclusively affected, not only those supplying the skin and the joints, but also the sensory nerves of the muscle. This peripheral degeneration is of course found most commonly in the legs, but the change in the nerves bears no relation to that in the spinal cord. The optic nerves when atrophied present wasting of the nerve fibres, and usually a remarkable increase in the interstitial connective tissue. Degeneration of the ascending or descending roots of the fifth nerve have been repeatedly found, and a degeneration of the intermedio-lateral tract and adjacent nerve fibres in the upper part of the cord, traceable up the medulla oblongata to the region of the so-called slender column, has been described in cases of the vasomotor and visceral symptoms. The sympathetic has been examined and found healthy, except in one case, in which the cervical sympathetic on the left side was degenerated in association with left exophthalmos.

In the diseased bones or joints conspicuous changes are found. There may be extraordinary wasting of the articular ends of the bones, which seems to begin by a rapid erosion of the cartilages. In the hip-joint, for instance, in a case recorded by Charcot, the edges of the acetabulum had been removed, and the whole of the head of the femur and most of the trochanter had disappeared. At the seat of old fractures, a large amount of bony callus is formed, and sometimes new bone is found in the capsules of the joints which have been injured.

Pathology.—The great fact in the pathology of tabes is, that it is a degeneration of the afferent nervous system, peripheral and central. The cerebral system suffers in far less degree than the spinal, but in the occasional peripheral degeneration of the optic nerve, and the central changes in the root of the fifth, we may trace the same law of distribution. The degeneration commences in the nerve elements themselves, the increase of connective tissue is secondary. This is true also of the disease of the peripheral nerves. There is every gradation, however, between the cases in which perivascular disease is great, and those in which it is so slight that there is nothing to suggest a primary interstitial process. It is important to remember that the overgrowth of neuroglia is a pathological process, distinguishable from degeneration of the nerve fibres that excites it. It may vary in degree in different cases, and, once excited, the energy of this tissue growth may be in some degree independent of its cause.

The chief causal fact regarding the disease is its common relation to syphilis. Tabes is generally, not invariably, a post-syphilitic disease. Certain elements of the nervous system degenerate, in consequence of the effect on them of previous or present syphilis. The usual symmetry of tabetic lesions indicates that the mechanism by which syphilis affects the nervous system is a blood state, and the manner in which the peripheral nerves suffer brings tabes into analogy with the more acute forms of peripheral neuritis, such as succeed acute specific diseases. The degenerative changes in the nervous system differ from the lesions of active syphilis, both in character and in time, in such a way as to make the assumption reasonable that they depend upon some product of the growth of the syphilitic organisms, a product which may possibly be a chemical substance, as was first suggested by Strümpell. Recent research is bringing into ever greater prominence the important part played by such products in the generation and course of disease.

Tabes is probably a rare sequel, regarded from the side of syphilis, but the occurrence of such consequences in some instances and not in others has analogies in the case of almost all the acute specific diseases that have similar sequelæ. The virus of the primary malady is no doubt organismal, and such variations are familiar to us in the case of other diseases. These considerations receive notable emphasis from the occurrence of tabes in children who are the subjects of inherited syphilis. The fact also shows how potent must be the part played by the agent compared with the susceptibility that is, as it were, the causal complement, a fact seen also in the rarity of cases of tabes in which syphilis can be absolutely excluded. The pathological facts already known enable us to understand many of the symptoms of the disease. There may be an interruption of the sensory path, either in the peripheral nerves or in the posterior fibres, and such an interruption in either place would explain the loss of sensibility which is so frequently present. The pains are probably due to the molecular changes in the nerve fibres, and their altered function. The long persistence of pain, without any increase in the symptoms, shows that they may be due to the action of structures that are changed but are not necessarily changing. The loss of reflex action from the skin is explained by the interruption of the sensory path, and the hyperæsthesia and increase of reflex action sometimes observed are accounted for by an increased sensibility of the sensory nerves. Whatever theory of the nature of the so-called tendon reflex action is held, the loss of the knee-jerk must be explained by an interruption of the sensory path; hence we can understand that this loss should be almost constant and early.

The mechanism of the muscular inco-ordination must be considered in the light of two fundamental facts. First, the ataxy cannot be primarily due to the loss of cutaneous sensibility, for complete anæsthesia of the skin may be present without the least ataxy. The second fact is, that ataxy may exist in considerable degree when the lesion is solely of the peripheral nerves. These two facts seem to show that the ataxy may be produced by one mechanism—the disease of the afferent muscle nerves. The posterior and median columns, extensively connected with the cerebellum in the direct cerebellar tract, probably conduct to the cerebellum the impressions from the sensory muscle nerves. If so, disease of these tracts and nerves must interfere with co-ordinating action of the cerebellum to cause some part of the loss of function which results from disease of the cerebellum itself. But there is another mechanism which may co-operate in increasing ataxy. The vertical fibres of the postero-external column have only a short course, and some probably connect the posterior grey matter at adjacent levels. Lesion of them may impair the association of the sensory structures, and so cause a want of harmony in the central mechanism. The varying character of the ataxy in different cases probably depends on local variations in the degree of change in the muscle nerves. Inco-ordination is probably chiefly an automatic process, depending on muscle reflex actions, and on the action of neighbouring sensory structures in the spinal cord, but chiefly depending on the function of the cerebellum, determined by the connection of the muscles with it; and the interruption of this connection is the chief element in the inco-ordination of locomotor ataxy.

Trophic changes in the skin, bones, or joints are probably connected with degeneration in the peripheral nerves, while the pains in the region of the fifth nerve are explained by the lesions of its root. Regarding the pathology of the visceral crises we know but little, but changes have been observed in the neighbourhood of the centre for the pneumogastric nerve, and this may be an indication of the causes of the disturbances that occur within the range of this particular nerve, especially of the gastric and laryngeal crises. The transient motor symptoms in the limbs and in the eyeball muscles are appparently of functional origin. They must be distinguished from the lasting palsies, which probably result from degeneration in the nerve and nuclei. But even the light reflex may return after it has been absent for years, and its loss must therefore depend, at least sometimes, on changes capable of recovery. It is important to remember that tabes is a disease which begins as a derangement of molecular nutrition. And if we remember that this derangement is the result of some toxic material circulating in the blood, we can understand better the variations in its course, its degree, and in the precise character of its symptoms.

Symptoms.—The typical symptoms are of three kinds—(1) motor, (2) sensory, (3) reflex; although all are ultimately traceable to the impairment of "sensory" impulses, using the term in its widest sense. (1) Inco-ordination of movement, commonly of the legs, occasionally of the arms also, without loss of power or muscular wasting; (2) pains, various in character, but especially sharp, momentary pains—"lightning pains"—in the affected parts, some impairment of sensation, very variable in degree and extent; (3) loss of reflex action, sometimes from the skin, and nearly always from the muscles, causing entire loss of the knee-jerk. There may be incontinence or retention of urine, and there is often loss of sexual power. Of these symptoms, the pain and the loss of the knee-jerk are by far the most common and earliest. They may exist alone, even for years. Other occasional symptoms are atrophy of the optic nerve, occasionally of other cranial nerves, trophic changes, especially in the bones and the joints, and curious paroxysmal visceral disturbances—gastric, laryngeal, rectal "crises," as they are termed. Actual motor paralysis and muscular wasting may occur as an extension of the morbid process to structures not affected in the common form.

Motor symptoms.—The ataxy develops gradually. At first it merely prevents perfect steadiness when the feet are close together, and especially when the eyes are closed. Another early symptom, which the patient perceives spontaneously, is the need for steadying himself against the washstand when he closes his eyes and bends forward over the basin.

As the defect progresses, the gait becomes changed, and there is difficulty in keeping the balance on turning quickly. As the inco-ordination increases, the change in the gait becomes greater; the feet are often raised too high, and brought down too suddenly. There may be little irregular movement of the legs, but not manifest general unsteadiness. Afterwards a stick is necessary, and the patient may be unable to walk without the help of another person. At last, even with help, it is impossible for him to stand, on account of the extreme irregularity of the action of the muscles. The inco-ordination is equally manifest when the patient is lying.

The arms may present similar inco-ordination, but they often escape. Occasionally they suffer before the legs. The affection is first revealed in delicate movements, and the patient becomes unable to button his coat. The grasp is not uniformly sustained, and if the hand is held out, especially with the eyes closed, there are slow, unintended movements of the fingers. The movements of the head, face, tongue, and eyes escape the characteristic derangement. Even with extreme inco-ordination, the power of the muscles may be unimpaired; but often some muscular weakness supervenes.

Sensory symptoms.—Spontaneous pains, especially those designated "lightning"—a name which sticks in the mind of every patient who hears it—are seldom absent. They are felt chiefly in the legs, not rarely in the trunk and arms, occasionally in the head. They occur in paroxysms, often at night, and last for hours or days at a time. Sometimes deep, they are often superficial, and then cause extreme hyperæsthesia at the spot where they are felt, and, indeed, trophic changes in the skin or hair may result. Less brief pains are often dull, or boring, or burning in character. Dull "rheumatic" pains also occur, and often early, and their nature is generally not recognised. In about half the cases, "gout" is the explanation. A painful sensation of constriction around the trunk—"girdle pain" —is not uncommon, and a similar sensation may be felt in the legs. Visceral pains also occur, but not frequently; they are usually paroxysmal in character, and associated with the disturbance of function above referred to. Other spontaneous sensations than pain are also common, such as tingling, "pins and needles," and a sensation as if the skin of the sole were covered by some soft substance is very frequent.

Diminution of sensibility is also present in most cases. It may involve all forms of sensibility, but those for pain and temperature may be impaired alone—seldom or never separately—or that for touch may be affected without the others.

There is often delay in the perception of pain, and also of heat and cold, which may amount to several seconds. Occasionally there is an after-pain. A prick in one spot, also, may be felt in other places (polyæsthesia), or a prick or touch on one leg may be referred to the other (allochiria). Sensibility is usually most impaired on the lower part of the legs and in the feet, especially on the soles. But, on the other hand, it may be perfect on the sole when it is lost elsewhere. It is often met with on the trunk in chief degree. It may involve the deeper tissues also, especially the muscles. Reflex action from the skin is usually impaired in proportion to the loss of cutaneous sensibility; in the early stage, however, it is occasionally increased. Loss of sexual power is a very common and often an early symptom, but it is not invariable; cases are met with in which the power persists to a late period of the disease.

Special senses.—In more than five-sixths of the cases of tabes, the reflex action of the iris to light is lost, contraction during accommodation being preserved, as was first noticed by Argyll Robertson. Often, also, the dilatation that occurs on painful stimulation of the skin of the neck can no longer be produced. Sometimes the ciliary muscle is also paralysed, causing loss of accommodation. The pupils are generally small, often very small; but they may be of medium size, or, very seldom, larger, and are frequently unequal. Paralysis of the external ocular muscles is also common, and may occur as transient weakness, lasting a few weeks, and then passing away; as permanent paralysis of a single nerve or part of a nerve; as combined palsy, suggesting a central origin; or as the progressive paralysis of many or all the muscles of both eyes—progressive ophthalmoplegia.

Atrophy of the optic nerve is the most serious ocular complication of tabes. Its frequency is difficult to ascertain, but is less than might be thought, certainly below 10 per cent. The ultimate tint is grey or whitish grey, the disc is hollow, and the vessels are not so narrowed as they are in atrophy after neuritis. The failure of sight usually commences with a peripheral limitation of the field, and loss of colour vision; acuity of sight often fails early, and in most cases the atrophy is slowly progressive, and usually ends in total, or almost total, blindness. It may, however, cease to progress, or may even undergo slight improvement. The atrophy is often more advanced in one eye than in the other, but very rarely does one only suffer.

Deafness is met with in some cases, sudden or gradual in onset, transient or lasting. Persistent deafness has been attributed to atrophy of the auditory nerve, but in many cases certainly without sufficient reason. Attacks of vertigo are frequent in the cases attended by deafness. Anosmia occasionally occurs, but the functions of the other cranial nerves are not often affected. Unilateral atrophy of the tongue has been sometimes noted; and in the larynx, more particularly in the cases in which laryngeal crises are present, paralysis or paresis of the abductor muscles of the vocal cords is not uncommon.

Sphincters.—The functions of the bladder are frequently deranged. There may be either sluggish micturition, or a tendency to incontinence; very often the bladder is not perfectly emptied, and the residual urine, unsuspected by the patient, gradually increases in amount, and the increased average resistance to the flow from the kidneys leads to their disease. Retention may be absolute, and may lead to overflow incontinence. There is rarely paralytic incontinence. Residual urine tends to decompose, and cystitis is apt to occur. The renal disease secondary to this is not an uncommon cause of chronic slight pyrexia, or of acute toxæmia and death, even when the conscious defect in micturition has been insignificant. The sphincter ani is also weak, so that a loose stool can be retained with difficulty; but its paralysis rarely reaches a high degree. Constipation is extremely common.

Vasomotor and trophic disturbances constitute an important group of symptoms. Ecchymoses and altered growth of hair have been known to occur in connection with sharp superficial pains. Herpes of the skin is not uncommon. Blisters may readily form beneath the epidermis of the sole, and may lead to indolent ulcers. Perforating ulcer of the foot has been found to be almost confined to cases of tabes. The nutrition of the nails of both the feet and the hands may be altered. The teeth, also, sometimes decay quickly, and may drop out. Changes in the nutrition of the joints and bones are, however, the most obtrusive effects. Charcot called special attention to it, and it is often called "Charcot's joint disease," also "tabetic arthropathy." Bones may become brittle, and this may lead to spontaneous fractures. Sometimes there is ossification of the adjacent structures, especially of the fibrous tissues connected with the muscles. It may follow an acute local inflammation. In the joints the changes consist of erosion of the cartilage, wasting of the ends of the bones, and ossification of the ligaments. With such changes, there may be effusion within the joints, and œdema outside of them. The larger joints are those most commonly affected, occasionally in the early pre-ataxic stage. Apart from these trophic changes, the knee-joint often becomes lax, so as to be bent backwards readily, in consequence of the variation in the nature of the support it should receive from the muscles, their contraction not being maintained, and the consequent overstrain of the ligaments.

Visceral symptoms may occur in many cases, usually paroxysmal, called crises. A frequent seat of such disturbance is the stomach; paroxysms of gastric pain are accompanied by vomiting, with or without nausea. Such an attack may last for hours or days, and recur in a few weeks or months. The chief intestinal disturbance is constipation; attacks of rectal pain, accompanied by distressing tenesmus, constitute rectal crises. In rare cases, attacks of pain have the character of renal colic, or sudden pains are felt in the neck of the bladder, or along the urethra, or at the meatus, with an intense desire to micturate—vesical or urethral crises. Laryngeal crises are not rare, but they vary much in character, so that their nature is often not recognised. The most common form is larnygeal abductor spasm, with noisy inspiration and expiration and dyspnœa. In other cases, cough is the most obtrusive symptom, coming on suddenly and distressingly severe, but without signs of spasm. These crises are sometimes, perhaps often, associated with weakness of the abductor muscles of the vocal cords, and the slightest noise during quiet breathing should lead to careful laryngoscopic examination.

Comparable to paroxysmal visceral symptoms, but extremely rare, are attacks of cerebral disturbance. They resemble those that occur in general paralysis of the insane, consisting of transient apoplectiform seizures, transient hemiplegia or convulsions, often unilateral.

Course and termination.—The course may be divided into three stages—(1) the preataxic stage, with pains, loss of the knee-jerk and of the iris reflex; (2) the ataxic stage, in which the patient is still able to walk alone, though there is distinct alteration of gait; and (3) the stage in which walking is possible only with the aid of another person, or is quite impossible. The course of the disease is very variable. In a far larger proportion of cases than was formerly conceived, the malady becomes stationary, especially in the early stage, and, when once stationary, it is not common for progress to take place in the future. When the symptoms increase, they vary much in rate; sensibility may fail, although inco-ordination does not increase, and conversely.

Periods in which the disease is almost stationary alternate with others in which the symptoms increase rapidly. Exacerbations may be distinctly excited by some influence such as a chill, a fall, or some alcoholic or sexual excess. There is nothing in the nature of the disease, in most cases, to produce death. The only direct effect of the malady which has ended life is laryngeal spasm or paralysis. Even the gastric symptoms, severe as they are, are never fatal. Death often results from intercurrent maladies, but many patients die from indirect results of the disease. Kidney complications are the most common. They often develop insidiously, and a patient still in the first stage may die from this cause after a few days' illness. Bedsores and pyæmia often cause death, but not so frequently as in most other spinal cord diseases.

Complications.—Tabes is sometimes complicated by true syphilitic lesions of the brain or cord, and acute or subacute myelitis may occur in the course of the disease. Other system diseases of the spinal cord may develop also as complications. The lateral columns may be diseased with the posterior, giving rise to ataxic paraplegia, but this is a distinct affection, and will be so described. Another complication is muscular atrophy, both the local atrophy already mentioned, and general atrophy, such as is met with in a pure spinal form. In rare cases the legs present the symptoms of tabes, while in the arms there are the signs of disseminated sclerosis. The most important and the most common complication of tabes is general paralysis of the insane. The two are often combined, and the symptoms of one or the other may predominate. It is sometimes said that the disease may commence as ataxy, and change to general paralysis, but it is probably more correct to say that there is a coexistence of the two affections, and the dominance of the symptoms of one or the other.

Of complications of tabes outside the nervous system, besides the indirect consequences of the malady, valvular heart disease is the most common. Coincident glycosuria occurring in tabes has been ascribed to an affection of the medulla, but it is extremely rare.

Diagnosis.—The diagnosis rests on the combination of the symptoms described. In the early stage, the loss of the knee-jerk, with pains and unsteadiness, justify the diagnosis of the commencing affection, providing we can exclude toxic peripheral neuritis, diabetes, diphtheritic palsy, and a lesion of the anterior horns or nerve roots. The diagnostic value of the loss of the knee-jerk can hardly be overrated. It is probably never absent in health, and in cases of disease in which the knee-jerk is present, the diagnosis of tabes is only justified by distinct and characteristic incoordination. In the few cases of this kind that have been observed, the knee-jerk has been usually unequal on the two sides, or has been entirely lost on one. The loss of the reflex is also of great diagnostic importance, but since it is not always lost in tabes, the negative significance of the normal reflex is far less than the positive significance of its loss. Its loss is, moreover, so often due to preceding syphilis, that it should always suggest this antecedent.

There are certain diseases with which tabes is especially liable to be confounded. One of these is multiple alcoholic neuritis, especially that variety which is known as alcoholic pseudo-tabes, in which the paralytic symptoms of the ordinary form of multiple neuritis may not be present. An altered electrical reaction may be found in the muscles, however, and there is generally marked muscular tenderness, and neither of those symptoms is present, as a rule, in tabes, nor are the pains of a "lightning" character, and the sphincters are not affected. The pupils act normally, and a history of alcoholism, as well as an improvement when alcohol is withdrawn, may help the diagnosis.

When the symptoms and lesions of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is really .one of the preponderance of symptoms of one or other malady, and in most cases the symptoms of general paralysis become more pronounced as time goes on, or the spinal symptoms pass into the background. All common forms of paraplegia are distinguished by the early loss of power, and the persistence, often increase of the knee-jerk, and the occurrence of a clonus. If weakness supervenes in tabes, it is usually late in the course of the disease, and if the knee-jerk has been once lost, it is not as a rule reproduced by secondary lesions of the cord. The same distinctions usually suffice to recognise the combined form of paralysis and inco-ordination which has been termed ataxic paraplegia. Acute lesions of the cord may produce inco-ordination and pain, but the suddenness of the onset and the limitation of the symptoms are generally sufficient to distinguish such a condition. Ataxy in one limb may also be produced by a tumour growing in the posterior column, but all such cases present the characters of a focal lesion with random consequences. In diphtheritic paralysis, in which the history of the sore throat is not clear, the loss of the knee-jerk may cause tabes to be suspected, but the nature of such a case is almost always rendered clear by the paralysis of accommodation and of the palate, which precede the ataxy. When the dorsal region of the cord is chiefly affected, the severity of the pains in the trunk may lead to suspicion of disease of the vertebral bones, but in the latter the pain is local and fixed, and is characteristically increased when the patient moves. The most frequent mistake is made in regard to the pains, which, especially when influenced by weather, are mistaken for rheumatism, or for sciatica when referred to the region of the sciatic nerve. The only organic intracranial disease with which tabes may be confounded, is tumour of the cerebellum. In some cases of tabes, the unsteadiness resembles that of cerebellar origin. Loss of the knee-jerk is strongly in favour of tabes, for, in cerebellar tumour, increase of jerk is the rule, and loss is only to be regarded as not incompatible. Anæsthesia and lightning pains are conclusive evidence of the spinal disorder, while, in cerebellar tumour, occipital headache is almost invariable, and considerable optic neuritis extremely common. The various visceral crises are not uncommonly mistaken for primary disorders of the deranged organs.

Prognosis.—Arrest of the disease is frequent, and considerable improvement is not rare. In the first stage the disease often becomes stationary. When ataxy is developed, the prospect of arrest is less than in the first stage, but is still considerable. When the third stage is reached, the likelihood of improvement is far less. On the other hand, in some cases the malady increases in spite of every effort, and even when there is permanent arrest, it is rare for all symptoms to disappear. The knee-jerk generally remains absent; often attacks of pain continue to occur. The fact of preceding syphilis has little influence on the prognosis. Only when the symptoms have developed rapidly, and the syphilis is recent, is there some ground to anticipate good from antisyphilitic treatment. In most cases the only trustworthy guide to prognosis is the observed tendency of the individual case. Diminution of the pain is a good sign, if other symptoms are stationary. It is often, however, a solace to the patient to know that the persistence of the pains does not mean progress in the disease. The optic nerve atrophy is usually progressive, but not invariably. Curiously enough, the occurrence of optic nerve atrophy makes the prognosis as to the spinal symptoms better. In most cases of atrophy the spinal lesion remains in the first stage.

Treatment.—In this disease treatment often has a distinct influence not only on the leading symptoms, but apparently also in determining the arrest of the disease. The first care should be to secure freedom from excessive mental work, anxiety, and physical fatigue. Absolute rest is often useful for a week or two at the commencement of the treatment. Care should be taken to avoid fatigue, risk of falls, and exposure to cold. A sea voyage may be useful in cases that are already stationary, but under other conditions a voyage often does harm rather than good. The digestive organs should be kept in careful order, and food should be light, wholesome, and easily digested. Alcoholic excess is especially harmful, and sexual excess is also most injurious. Smoking should be either avoided, or restricted to small amount.

The influence of drugs on the disease is unquestionable. There is no specific for the malady, and the influence of drugs is variable, that which seems to do much good in one case having no influence on another. The influence of antisyphilitic agents is distinct only in rare cases, chiefly in cases in which the symptoms have developed rapidly, and the syphilis is recent. But so widely spread is the opinion that every consequence of syphilis is amenable to treatment, that it is often well to avoid the suspicion of neglect, and even to clear the therapeutic ground by a course of iodide of potassium for five or six weeks. Very small doses of mercury may also be given for a longer time, and may often be conveniently combined with other drugs. It seems sometimes to have a tonic influence, but in late cases, and in all the late nerve degenerations that follow syphilis, energetic mercurial treatment only does harm. The drugs which are most useful in tabes are arsenic, iron, quinine, aluminium, nux vomica, strychnine, nitrate of silver, Calabar bean, belladonna, ergot, and phosphorus. Arsenic and chloride of aluminium are certainly those which most frequently do distinct good, and they may be given in combination, the chloride of aluminium in doses of 2 to 4 gr., three times a day. Counter-irritation of the spine, most safely effected by the thermo-cautery, is sometimes useful, chiefly in cases in which there is a tendency for the symptoms to increase, or in which there is spinal pain or tenderness. It probably acts by lessening any secondary inflammation. Of special symptoms requiring treatment, pains are the most important. The most useful agents for relieving paroxysms are antipyrin, acetanilide, or phenacetin. Hypodermic injections of cocaine generally arrest for hours pains that are superficial in character. The quantity injected should be, in the first instance, not more than $\frac{1}{12}$ gr., but may be increased to $\frac{1}{6}$ or even $\frac{1}{4}$. Superficial pain is also relieved by local application of chloroform, and occasionally the pains are lessened by a warm bath. Paroxysmal visceral symptoms often yield to one or other of the agents which relieve the pains, but when severe, morphine alone mitigates their intensity. Slight gastric crises may be relieved by sinapisms to the epigastrium and the neck. Nitrite of amyl and nitroglycerin may relieve laryngeal spasm. Weakness of the bladder is sometimes lessened by nux vomica or strychnine, and the latter, given hypodermically, seems to have marked influence in certain cases. Nocturnal passage of urine is occasionally arrested by belladonna. Blisters or sores on the feet should receive careful local treatment, and the patient should be cautioned never to cut a corn, as a perforating ulcer is not infrequently set up by a corn being cut too deeply. Attention to the bladder is most important. If there is any reason to believe that the bladder is imperfectly emptied, a catheter should be passed, and if residual urine is found to be present, a catheter should be used systematically, and the bladder washed out every day or every other day. Death occurs, in many instances, from the consequence of unsuspected retention. The employment of systematic movements of the limbs, as elaborated by Fraenkel, is of undeniable utility. Electricity has little influence on the chief symptoms of tabes. Faradisation of the skin may occasionally be useful for relieving the defect of cutaneous sensibility; and faradisation to the epigastrium or perineum may occasionally be useful when the bladder or its sphincter is weak. Nerve stretching, which was fashionable a few years ago, is now never used in the treatment of tabes. Treatment by suspension, more recently introduced, may be said to be similarly obsolete.

HEREDITARY ATAXIA.

Syn., Friedreich's Ataxy.

This disease, known also as hereditary ataxy, is named after the Heidelberg professor who first described it in 1861. In some cases which came under his notice he recognised a close resemblance to the condition then recently described by Duchenne under the name of locomotor ataxy. These cases, however, differed particularly in that there was evidence of the presence of the same condition in more than one member of the family, and that there was an absence of any sensory impairment.

Etiology.—The important point is the tendency manifest in the great majority of instances for the affection to attack more than one member of the same family, Alcoholic over-indulgence and syphilis in the parents have each been invoked as possible factors in the causation, and the evidence of the presence of one or other of these conditions, sometimes of both, has been in many instances indisputable. On the other hand, there is nothing like conclusive proof of the constant presence of either or both. Two particular diseases, diphtheria and smallpox, have been supposed to be peculiarly efficacious in evoking the early symptoms.

Pathology and morbid anatomy.—The characteristic lesion found post-mortem has been a sclerosis, widespread, affecting especially the posterior columns, but in a less degree also the lateral and anterior. The cells in Clarke's column have also been found atrophic. But a peculiar smallness of the spinal cord has also been noticed in several of the cases, a condition noted by Friedreich himself. Besides this evidence of defective development in the spinal cord, it has also been observed that the cerebellum in some cases was not so well developed, the condition supposed to underlie the manifestations of Marie's hereditary cerebellar ataxy. In one of Menzel's cases there was degeneration of the posterior columns, of the lateral and anterior, but there was also degeneration of the hypoglossal and facial nuclei, and atrophy of the cerebellum, pons, and olivary bodies. The atrophy of the cerebellum was associated with a deficiency of the cells of Purkinje. A case similar in its clinical aspects to that of Menzel, and almost identical as regards the cerebellar changes, the cord showing no alteration, was described several years ago by Fraser and Coats of Glasgow. This case may be regarded as a connecting link between these two types, and whether future observation will be able to bridge over whatever small gulf remains between those cases and cases of Friedreich's ataxy, it is impossible to say. It seems likely, however, that at no very distant time it may be possible to demonstrate that all belong to a group of cases in which there is defective development, and consequent liability to early decay, of the nervous system, especially on its afferent side, and that the slight differences between them are dependent upon the defect being manifested in different degrees in different parts of the same system.

Symptoms.—The onset is usually marked by difficulty and uncertainty in walking. In standing, the feet are placed widely apart and the eyes are fixed on the ground, the attitude imparting to the patient an aspect of pre-occupation. This difficulty usually commences before 10, and gradually becomes greater, until at length the patient is unable to walk without support, finally unable to walk at all, or even to stand. With the increasing difficulty in walking, there is also a loss of rhythm and regularity in utterance and phonation. The final syllables in words are clipped, and a certain explosive character is imparted to the articulation. Speech proper does not suffer, and intelligence may remain unimpaired. The movements of the hands also become coarsely tremulous and uncertain, but the lightning pains so characteristic of tabes are never present, although slight cramplike pains, or even actual cramp, is occasionally complained of. The sphincters are unimpaired, except for occasional slight weakness, and there is no sensory affection or trophic disturbance. With difficulty in walking there is frequently associated development of deformity in the foot, *pes cavus*, and also of lateral curvature. The pupillary reaction is not interfered with, and there is no optic atrophy. The deep reflexes are abolished, not only the knee-jerk, but also the wrist and elbow-jerks. Occasionally, however, in cases of Friedreich's ataxy, otherwise characteristic, the knee-jerk has been found present. Examination of the heart usually reveals the presence of a distinct murmur. A few years ago, from a consideration of several different cases, Marie concluded that there exists a type of disease in many points resembling Friedreich's ataxy. This he named hereditary cerebellar ataxy, and he regarded the symptoms as the result of some congenital defect in the cerebellum. The ataxy, the articulatory difficulty, the frequent presence of nystagmus, and the fact that more than one member of the family might suffer, suggested a close resemblance to Friedreich's disease, yet the occasional presence of the Argyll Robertson pupil, the not unusual occurrence of optic atrophy, and the invariable exaggeration of the knee-jerk, form important points of distinction. The onset of symptoms also in this disease was usually at a much later date than in Friedreich's ataxy. A remarkable series of cases described by Sanger Brown probably belongs to this class. Nonne and Menzel have also described similar conditions, but in Nonne's case ocular movements were impaired, speech was loud, nasal, and explosive, and there was mental weakness. Other observers have described cases of hereditary infantile spastic paraplegia, in which the symptoms are merely those of lateral sclerosis, and there is neither the ataxy nor the deformities which are described in association with Friedreich's disease.

Diagnosis.—To any one unfamiliar with the disease, the diagnosis is difficult. The early age, the absence of characteristic pain, and of the affection of the pupil, suffice to distinguish the disease from ordinary tabes dorsalis. In many points it presents a close resemblance to the ataxic paraplegia of Gowers, but in the latter also the age at which the symptoms commence is usually much more advanced than it is in cases of Friedreich's ataxy. Disseminated sclerosis offers some resemblances, but the exaggeration of the reflexes and the absence of deformities are usually sufficient to distinguish the two. Reliance must, as a rule, be placed on the general picture presented, and attention in any doubtful case particularly directed to the family history, the state of the deep reflexes, and the presence or absence of deformities.

Prognosis.—This is necessarily bad. There is no immediate danger to life, the patients often live for many years after they become completely helpless; but arrest of the process, far less improvement, is, with our present knowledge and resources, scarcely to be expected.

Treatment.—This chiefly cousists in attention to general health and to the symptoms as they arise. The anæmia is usually benefited by the administration of iron and arsenic, and there is no contra-indication to strychnine, which often does good, by improving the patient's appetite and general condition. The cramps are usually not so severe as to call for any treatment except gentle rubbing. It is doubtful if much good is effected by dividing the tendons of the muscles whose contraction produces the foot deformity.

ATAXIC PARAPLEGIA.

This seems the most accurate designation for a disease of the spinal cord which presents a combination of the symptoms of paraplegia and ataxy. It consists of combined changes of the posterior and lateral columns.

Etiology.—Neurotic heredity can be traced in about one-tenth of the cases. A history of syphilis is rare. The disease usually commences between 30 and 40, and affects males more than females. Exposure to cold and severe exertion have been apparent exciting causes, and a severe concussion of the spine and great sexual excess have preceded the symptoms by a few months. In many cases no distinct cause can be discovered.

Morbid anatomy.—In all cases the spinal cord has presented sclerosis of both posterior and lateral columns, usually less intense in the lumbar than in the dorsal region. In rare cases the whole posterior column in the lumbar region is diseased, but that part of the external column near the commissure, and near the neck of the posterior horn, usually remains free. When the degeneration is considerable, the posterior median columns in the upper part of the cord may present the usual ascending degeneration of secondary origin.

The degeneration in the lateral columns is also variable in extent and position, and may not be strictly limited to a single system of fibres. The pyramidal tracts, however, are chiefly affected, but the mixed zone of the lateral columns, the lateral limiting layer, and even the direct cerebellar tract, may be invaded. The direct pyramidal tract may be affected as well as the lateral, and, as in most other degenerative diseases of the cord, there may be some increase of connective tissue in the unaffected columns. As a rule, the membranes and the grey matter have appeared normal, and the muscles have been found normal.

Pathology.—The double lesion in the posterior and lateral columns supplies an explanation of the symptoms which characterise the disease. The fact that the lumbar root zone is unaffected accounts for the integrity of the muscle reflex action. The only recognisable lesion that can be regarded as the cause of the paralysis is the degeneration of the pyramidal tract. The variations in the amount of disease, which does not always correspond to the degree of palsy, may be caused by degeneration of the termination of the fibres, which may be much greater than that of the fibres higher up the cord. The chief pathological difficulty arises from the fact that the morbid process may extend beyond the limit of the tracts chiefly diseased. Its tendency to assume this diffuse character has led some observers to regard it rather as a chronic myelitis than as a system of degeneration. It may occupy an intermediate position. Sometimes the primary morbid tendency may be doubled, involving both the nerve elements and the interstitial tissue.

Symptoms.—The symptoms are usually low in onset, and in the early stage resemble those of spastic paraplegia, with the addition of unsteadiness. The legs suffer first, and often alone; the weakness may be slight, is often greatest in the flexors of the knee and hip, but frequently more in one leg than in the other. The unsteadiness is distinct when the feet are placed close together, and inco-ordination also is revealed by the patient's gait. Lightning pains are almost always absent. Dull pain in the sacral region of the spine in often an early symptom. Girdle pain is rare, and as a rule there is no impairment of sensibility. Reflex action from the sole may be normal or increased, but the cremasteric and abdominal reflexes are sometimes lost. The knee-jerk is quick and extensive, and there is generally a distinct rectus clonus. The foot clonus is also commonly to be obtained, and if the arms are affected, the myotatic excess is shown in those limbs also. There may be conspicuous ataxy of the hands, and a tendency to cramp-like spasm on an attempt to use them. Sexual power is often early lost in the disease; the sphincters may be impaired, and inability to empty the bladder is apt to develop insidiously. The iris usually acts normally, but occasionally the light reflex is lost while accommodation persists, and, on the other hand, accommodation may be lost while a normal reaction to light remains. Optic nerve atrophy is rare, and the external ocular muscles are, as a rule, unaffected, except that nystagmus is frequently present on movement of the eyes. Slight articulatory impairment is not uncommon, and there are occasionally tremulous movements of the face, resembling those of general paralysis. With increasing weakness, the aspect of the patient comes to be that of spastic paraplegia. The motor weakness may go on to complete paralysis. In spite of the progress of the weakness, sensation remains unimpaired, and the cranial nerves do not suffer. Among complications, the most important are mental changes, like those of general paralysis, slight muscular atrophy, and arthritis of doubtful significance.

Diagnosis and prognosis.—The condition of the knee-jerk distinguishes it sufficiently from tabes, and there is not the loss of power in that disease which is found in ataxic paraplegia. The resemblance to primary spastic paraplegia is close, but the presence of inco-ordination, or a history of it, is usually a sufficient distinction. In cases in which this may be slight, the diagnosis may be difficult. So-called hereditary ataxy presents a close resemblance to this disease, and is probably intermediate between it and true tabes. It is distinguished by its occurrence in several members of a family, and by the loss of the knee-jerk, as well as by the presence of nystagmus and the characteristic impairment of articulation. The distinction from diffuse or focal chronic myelitis will be evident from what has been said in reference to the morbid anatomy. A tumour in the middle lobe of the cerebellum may cause symptoms similar to those of ataxic paraplegia; but the weakness in the legs is never great in cerebellar tumour, and occipital headache vomiting and optic neuritis are present in most cases. In ataxic paraplegia this is similar to that in the spastic form, and depends on similar considerations, which need not be repeated.

Treatment.—Treatment of ataxic paraplegia is the same as that of allied diseases, and the reader is referred to the section on treatment in the article on Spastic Paraplegia.

SCLEROSIS FROM TOXIC BLOOD STATES.

Toxic blood states have recently been recognised as the cause of sclerosis of the cord. Diabetes has been shown by Williamson to be occasionally associated with posterior sclerosis. Lichtheim and others have pointed out the occurrence of widespread sclerosis in association with pernicious anæmia and analogous conditions, and Putnam has demonstrated the occurrence of similar sclerosis in connection with enfeeblement in persons past middle life.

Morbid anatomy and pathology.—There is usually widespread sclerosis in the lateral, anterior, and posterior columns, most marked in the cervical region, least marked in the lumbar, and not affecting any except the lowest part of the medulla. The sclerosis apparently consists in a parenchymatous degeneration of the nerve elements and consequent thickening and increase of the neuroglia. There is a marked increase in the number of vessels in the affected region, but no definite infiltration of the surrounding tissue. It may be that in some cases sclerotic foci may exist, the result of hæmorrhage from the vessels whose walls are altered, usually thickened. The cause of the sclerosis is probably some toxic blood condition. The changes in pellagra, an admittedly toxic disease, are similar in character and distribution. Sclerotic foci, as a result of hæmorrhages, may account for the occasional departure from true symmetry, of the sclerosis which is found in these cases. All the appearances, however, point to the presence of some toxic blood state as the intimate cause. That it is not myelitic in nature is shown by the absence of degenerative changes, secondary to foci of inflammation, and by the absence of exudation.

Symptoms.—Difficulty and uncertainty in walking usually succeed a period of bad health. Sharp pains occasionally occur, the knee-jerks at first are normal or diminished and may differ on the two sides, occasionally they are early lost, sometimes exaggerated. The weakness gradually increases, and sensory impairment may occur. There may be marked rigidity, and the arms may be affected. The signs of pernicious anæmia—enlarged spleen, altered blood, etc.—are not a necessary accompaniment of the condition, which usually ends fatally through exhaustion, or it may be by the secondary effects of cystitis.

Diagnosis and prognosis.—The cases occasionally resemble conditions of toxic peripheral neuritis. The most frequent resemblance, however, is to ataxic paraplegia, and undoubtedly some cases described as ataxic paraplegia have been of the nature of the cases under notice. Occasionally, in cases in which the knee-jerk is lost, there is a close resemblance to tabes dorsalis. There is, however, no pupil change, and affection of the sphincters, if it occur, is late, and the pains are not characteristic. The prognosis is unfortunately bad. In some cases, however, there has been at least temporary improvement, and it is possible that occasionally this might become permanent. This is particularly the case if anæmia is obtrusive.

Treatment.—Attention to general nutrition is necessary; iron and arsenic are useful, if there is distinct anæmia. Bedsores, cystitis, and other complications must be treated *secundum artem*, and pain must be relieved, if necessary, by opium or morphine.

SYRINGOMYELIA.

Etiology and morbid anatomy.—Cavities in the spinal cord are met with at all ages, and those which present certain features or cause certain symptoms are now known by the above term. The term is applied to all cavities in the cord with well-defined limits, but the majority of these are surrounded by a layer of embryonal neuroglial tissue, and outside these the white substance of the cord is deficient in amount. Wherever met with, such a condition stamps the cavity as congenital, because this normal material is due to a persistence of the embryonal tissue from which the cord is developed. Moreover, the cord sometimes suffers serious damage from pressure, owing to the distension of the cavity by the liquid which accumulates within it, or by the growth of adjacent tissue. The condition is not rare, but is often overlooked, especially in its slighter forms. The term hydromyelia is applied to the simpler forms, in which the cavity is merely a dilated central canal. But as there is no real difference between this variety and others, the term is unnecessary and even misleading. In the process of development of the cord, the central canal ultimately lies in the anterior part or middle of the grey commissure. It may persist through life as a cavity, or as a slit lined with epithelium. Around the canal there are usually many nuclear elements, and these frequently fill up the lumen of the canal, so that its position may be indicated only by a mass of cellular elements. The obliteration of the canal often occurs early in life, and is no evidence of any morbid process. It may also be found obliterated in one part of the cord, and patent in another. Syringomyelia occurs chiefly in the cervical and upper dorsal regions of the cord. Occasionally the cavity exists through the whole length of the cord. In the simple condition, the morbid change presents the two features already mentioned—a quantity of tissue, translucent in aspect, persistent embryonal tissue, and a cavity due to imperfect contraction of the canal. This change is always confined to the posterior half of the cord, and usually to the posterior columns, which, in the process of development, are the last to be formed. When a persistent tissue is considerable in quantity, breaking down may occur, and so a new cavity arise, or an enlargement of the primary one. It will thus be seen that the cavity may be distinct from the canal, and in some cases the abnormal tissue around it is very small in amount.

The cavities in the adult cord present many varieties. The most common form resembles, in its essential features, the congenital condition, and it is practically certain that the two forms are really the same, and that the form met with in adults is the congenital condition persisting until later life. The chief difference lies in an apparent increase of the gliomatous tissue, by a process of growth, and the consequently greater damage to the other parts of the cord by the distension of the cavity. This damage involves most the nearer and less resisting parts. The grey substance therefore suffers in greatest degree, the white columns to a less but variable extent. As the simplest condition, we may have a dilatation of the central canal, surrounded by gliomatous tissue, disposed as in congenital cases. This tissue is often more abundant, and forms a more distinct mass lower down the cord, below the cavity.

It has been pointed out that in the congenital cases a process of growth must take place in the persistent embryonal tissue, to account for some of the conditions met with. Hence it is not surprising that in many cases the growth should attain the dimensions and assume the characters of a positive tumour. In most cases the tumour has been central in position, and had the structure of a glioma. It has occupied a large portion of the area of the cord at a certain part, and it is not surprising that the condition of syringomyelia, even if congenital in origin, should be frequently associated with definite tumours, more especially since, as has been pointed out elsewhere, sarcomata may grow from the tissue around the central canal.

The posterior cornu is not an uncommon seat of cavities, which may extend through the whole length of the horn; and, in one case of such a cavity, hæmorrhage occurred into the cervical region of the cord, and the cavity referred to in the posterior horn was filled with blood throughout the entire length of the cord. Such a condition suggests the possibility of certain obscure cases of spinal cord disease being of this nature, namely, hæmorrhage into a congenital cavity.

In the conditions referred to, definite limited regions of the cord have been involved through a considerable vertical extent. The changes are, however, much more diffuse, and we may have, scattered through the nerve substance, tracts of peculiar tissue associated with cavities mostly like fissures, the whole appearance suggesting an active and recent morbid process. Yet even in those cases the probability is that the origin of the condition is congenital; and one point in favour of such a view is the fact that the morbid changes, as in other forms of syringomyelia, are chiefly in the posterior half of the cord, and that the minute structure of the abnormal tissue is exactly like that of the normal gelatinous layer beneath the pia mater.

The damage to the grey matter may entail alterations in the related nerves, and even in cases of long duration, in those connected with the posterior root. In the peripheral nerve fibres of the skin and muscles, changes of the ordinary chronic degenerative character have been found in cases in which sensation was altered or muscles wasted.

Thus these forms of syringomyelia, various as they are in seat and form and general aspect, seem all to be connected with gliomatous tissue, but we are still ignorant of the mechanism by which the defect of the process of development is brought about. Inflammation of the embryonal neuroglia at an early stage of development has been supposed to be the cause, but of this there is no real evidence. It is possible that more than one morbid process during the early stage of development may cause the condition, and that closure of the canal below may have consequences during development which it has not at a later period. But it is important to recognise the fact that there is a gradation between the varieties, and that some are associated with developmental defects in the brain, to which they can only be related as the common expression of a defect in the developmental tendency of the germinal tissue. And what must be true of some forms is probably true of most. The symptoms of the disease have followed some illness, mental shock, anxiety, or traumatic influence. But it is probable that such conditions have only been the means of evoking the manifestations of pre-existing disease.

Symptoms.—Sometimes there is no history of symptoms in cases in which the condition has been discovered after death. In young children, indeed, it appears never to cause any definite symptoms, when the disturbance of function is variable, both in its incidence and in its degree. The fact that the lesion is almost always greater at the cervical region, causes the arms to be the most common seat of its manifestations; while the invasion of the fourth ventricle by the associated growth explains the involvement of some of the cranial nerves that has been met with in a few cases. The symptoms usually are slow in development, and the two leading features are loss of sensibility—chiefly to pain and temperature—and muscular atrophy. The sensory loss is the earlier and more constant. Muscular atrophy is the result of damage to the anterior horn; and destruction of the motor nerve cells involves the degeneration of the nerves and the wasting of the muscles. The course of thermal sensations is still quite unknown, but the only fact we have—the frequency with which such sensibility and sensibility to pain are involved together, points to contiguity of path, and prevents surprise at the special affection of this form of sensibility in syringomyelia. Tactile sensation is lost only in rare cases. Perversion has been noted, heat being felt as cold, and *vice versâ.*

Spontaneous sensations are common, doubtless from the irritation of the fibres. Pain in the spines may be complained of, and is probably a direct effect of the disease. The loss of sensation is usually sharply limited, and is as a rule found in the same part as the muscular wasting, but more extensive. The muscular sense is said to have been generally normal when tested.

The motor symptoms usually succeed the sensory loss. When the legs suffer, it is generally from simple spastic paralysis. The arms are the chief seat of the muscular wasting. Rarely there is atrophy of the legs, similar to that in the arms, and in such cases the disease extends into the lumbar enlargement. Reflex action in the legs varies according to the state, the knee-jerks being usually increased, and foot clonus being occasionally present. Weakness of the trunk muscles often leads to lateral curvature of the spine, convexity being usually to the left. Both sides usually suffer together; a unilateral affection has been observed, but is altogether exceptional. The sphincters may escape, or they may be involved. Trophic disturbances in the extremities are not uncommon. The skin may become thin and glossy, or thick and horny. The nails share in these changes, becoming blue, fissured, and cracked; they may even drop off. The secretion of sweat has been lessened in the part, as has been noted definitely in one unilateral case. The bones may become thickened or brittle, and joint changes, such as those in tabes, may sometimes occur.

Occasionally, disturbances of function in the parts supplied by the cranial nerves have been noted, the result presumably of extension of the changes in the nervous system into the medulla and pons. Thus there has been paralysis of one vocal cord, of the tongue and of the face, difficulty in swallowing, disorder of respiration and of cardiac action. The pupils are often unequal; narrowing of the palpebral fissure, and slight ptosis have also been noted, and nystagmus is not rare. The course of the disease is slow, and in most cases death is the result of exhaustion, and the impairment of the functions of the medulla, or, more commonly, of some complication, such as bedsores, cystitis, and the like.

Diagnosis. — Cervical pachymeningitis causes symptoms which, in general character and in distribution, very closely resemble those of syringomyelia, but the affection runs a more rapid course; anæsthesia involves all forms of sensibility, and is not of greater but of less extent than the muscular wasting. Pain, also, is a far more prominent symptom, and the reaction of degeneration is common in wasting muscles. Tumours of the spinal cord may also cause similar symptoms, but the resemblance is often more than superficial, for such growths generally arise from residual embryonal tissue, and are such as are frequently associated with syringomyelia. It is chiefly from the more rapid development of symptoms that such a growth can be suspected. Progressive muscular atrophy is sufficiently distinguished by the absence of sensory impairment. Hæmorrhage into the cord is the only acute affection which enters into the diagnostic problem, and the presence of a cavity into which blood has been effused may be suspected, if the hæmorrhage has been preceded by any symptoms suggestive of syringomyelia; or if a rapid extension occurs through a considerable vertical extent of the cord, distinctly abolishing its central function. A peculiar difficulty in diagnosis is presented by the cases, which are not few, in which the first symptoms follow an illness, a blow, or even mental disturbance, which seems to be their cause, and suggests disease of a very different nature. In such cases, waiting and watching are the only means of arriving at a right opinion.

Prognosis and treatment.—The prognosis and treatment of this condition are, unfortunately, subjects on which its nature permits little to be said. When the central functions of the cord are considerably impaired, it is rare for life to be prolonged more than one or two years. On the other hand, a stationary condition may last for many years; but treatment can do nothing for the morbid state; nor is it likely that the progress of spinal surgery will afford the means of relief. But trophic changes may be diminished by care and early treatment. Pain may need the customary sedatives, and early pain, especially the hyperæsthesia depending upon the intensification of nerve impulses at the irritated part, may be much lessened by the application of cocaine, used as in tabes dorsalis.

MORVAN'S DISEASE.

Syn., Panaritum Analgicum.

This is the name usually applied to a condition which was described by a physician of Brittany in 1883. Observations are not numerous, but there seems little doubt that the disease is essentially a form of syringomyelia, although it may be said to consist essentially in a combination of that condition with peripheral neuritis in the extremities. The characteristic symptoms are in the upper extremities. Neuralgic pains may occur at first, with subsequent weakness and muscular wasting, with loss of all forms of sensation, and especially a peculiar trophic change, whitlows on the fingers, with recurring deep ulcerations in various parts of the digits; these changes occurring without producing any pain. Occasionally the trophic disturbance precedes the anæsthesia, in which case the former is accompanied by pain. Vasomotor derangements, lividity, pallor, etc., often precede and accompany the disturbance of nutrition. The loss of sensation is often more extensive than the trophic changes. The feet are occasionally, though rarely, the seat of anæsthetic ulceration, and the legs have been found weak, with excessive knee-jerk and foot clonus. Anæsthetic leprosy presents the closest resemblance to this disease, but the peculiar ulcerations are absent, and pigmentary alterations occur in the skin. In Raynaud's disease, there is not the peculiar loss of sensibility, nor an equal tendency to whitlows, and vasomotor disturbance is more conspicuous. In the peculiar malady known as sclerodactyla, there is no loss of sensibility, nor tendency to destruction of the finger ends, and simple syringomyelia is distinguished by the common absence of whitlows, although, as we have said, there is reason to believe Morvan's disease is really a variety of syringomyelia.

SPINA BIFIDA.

This depends upon a defect in the closure of the vertebral arches, leading to the protrusion of the membrane as a sac, into which the lower part of the spinal cord often extends. The condition is most common in the lumbar region of the spine, and is said to occur in about one out of every thousand children born. In many cases the central canal of the cord is enlarged above, and the lower part may be similarly distended. The essential element in spina bifida—the defect in the vertebral arches—depends on a deficiency in the ingrowth of the mesoblast, which should enclose the embryonal spinal cord, and form bone. A similar growth of mesoblast forms the chorion, muscle, etc., and this apparently takes places in excess in spina bifida occulta, and leads to a curious condition usually present, an excessive growth of hair over and about the affected part. On the other hand, in many cases in which there is a tumour, this superficial mesoblastic ingrowth seems to be deficient, so that at the upper part of the tumour there is an area in which there is no proper skin, but a peculiar, shiny membrane, destitute of hair. This deficiency probably permits the protrusion and formation of an external tumour. Of the several forms, that without an external tumour (spina bifida occulta) has been least frequently met with, although it is probable that a knowledge of the significance of the growth of hair in the lumbar enlargement would lead to the detection of this state in many more cases. Putting this form aside, simple meningocele and syringomyelocele are both rare, the common form being meningomyelocele, in which the cord, altered or intact, extends within the sac. The lower part of the cord is generally adherent to the posterior wall of the sac, where its traction often causes a depression in the surface.

As a rule, the central canal of the cord is not continuous with the sac. Ordinary syringomyelia may exist in the upper part of the cord, as has been already stated. The extension of the cord in the wall of the sac probably indicates a developmental defect similar to that of the bony canal. In rare cases there has been no proper development of the cord: but in one case it was represented by mere nerve-like string, in a child who also had hydrocephalus, but lived five weeks.

Symptoms may be absent if the cord is intact. In other cases, where there is defective development of the lower part of the cord, this is manifest by an absence of function, amounting even to complete paraplegia. In slighter forms, the paralysis and wasting may be partial, and some children, whose legs have appeared natural, have never been able to stand, the result possibly of a defect in the posterior columns. Even in the slighter forms, paralysis and atrophy have come on after some years. Indeed, in the unsuspected forms without a tumour, there may be special danger of injury to the unprotected structures, and of damage to nerve roots. The muscular paralysis and atrophy have usually been below the knees, and the anterior tibial muscles are the most prone to suffer. Hence the most common form of deformity is talipes varus. Chronic changes in the skin of the feet, and in the bones of the tarsus, and of the metatarsal bones, have also been met with. These symptoms may be greater on one side, and may be a result of the greater degree of damage to the posterior roots, and perhaps of descending neuritis.

The treatment of spina bifida is purely surgical, and that of its consequences is the same as for similar conditions in other diseases.

W. R. GOWERS.
JAMES TAYLOR.

DISEASES OF THE BRAIN.

REGIONAL DIAGNOSIS.

THE diagnosis of the seat of a lesion depends upon an accurate knowledge of the structure and functions of the brain. It is not proposed here to do more than point out some of the more important facts relating to—(1) the vascular supply of the brain; (2) the functions of the several parts of the brain; (3) the special symptoms, paralytic or otherwise, which may serve to localise the lesion.

Arterial supply.—The brain is supplied with blood by the two vertebrals and the two internal carotids; these two systems are connected at the base of the brain to form the circle of Willis. This is a provision whereby occlusion of one of the principal arterial trunks mentioned does not lead to the deprivation of any portion of the brain of blood. The circle of Willis is formed by the basilar dividing into the two posterior cerebrals, and these are connected on either side with the internal carotids by the posterior communicating. The internal carotids severally divide into the anterior and middle cerebral arteries; the two former are connected in front by the anterior communicating, thus completing the circle.

The arteries of the brain may be divided into three systems—cortical, basal, and basilar and vertebral.

Cortical arteries.—These are the anterior, middle, and posterior cerebral, the two former are the result of division of the internal carotids, the latter of the bifurcation of the basilar. These three arteries are distributed over the whole surface of the brain (Figs. 14, 15, and 16). Each main trunk gives off from two to five secondary branches, and these again divide several times in the pia mater before entering the brain substance. The nutrient arteries of the cortical system are slender and delicate, and are of two varieties—short, terminating in the grey matter of the cortex; and long, passing through to the subjacent white matter of the centrum ovale. Since there is only a very limited and variable anastomosis between the cortical arteries and their branches and terminal ramifications, occlusion by a thrombus of a branch of the main arterial trunk will cause a patch of softening of the grey matter and subjacent white matter; the size of the softened area depending upon the size of the vessel and degree of anastomosis with other vessels.

More precise details will now be given of the distribution of the cortical arteries. A glance at Figs. 14, 15, and 16 shows that the most important, as regards area of supply, is the middle cerebral; moreover, it is much more likely to be the seat of embolic occlusion, owing to its being the main continuation of the internal carotid. It is also more often affected by inflammatory and degenerative changes than the other two arteries; consequently it and its branches are more often the seat of thrombosis. As it supplies most of the centres having special functions, particularly those connected with voluntary movements in the opposite half of the body, a common result of occlusion of this vessel or one of its branches is hemiplegia or a monoplegia. Each middle cerebral enters the fissure of Sylvius, and after giving off the lenticulo-striate branches, and a branch to the uncus, passes over the floor of the island of Reil, and then divides into five branches, the distribution of which is indicated in Fig. 13. It will be observed by reference to Figs. 14, 15, and 16 that the area supplied by the middle cerebral includes portions of the second and third frontal, Broca's convolution,

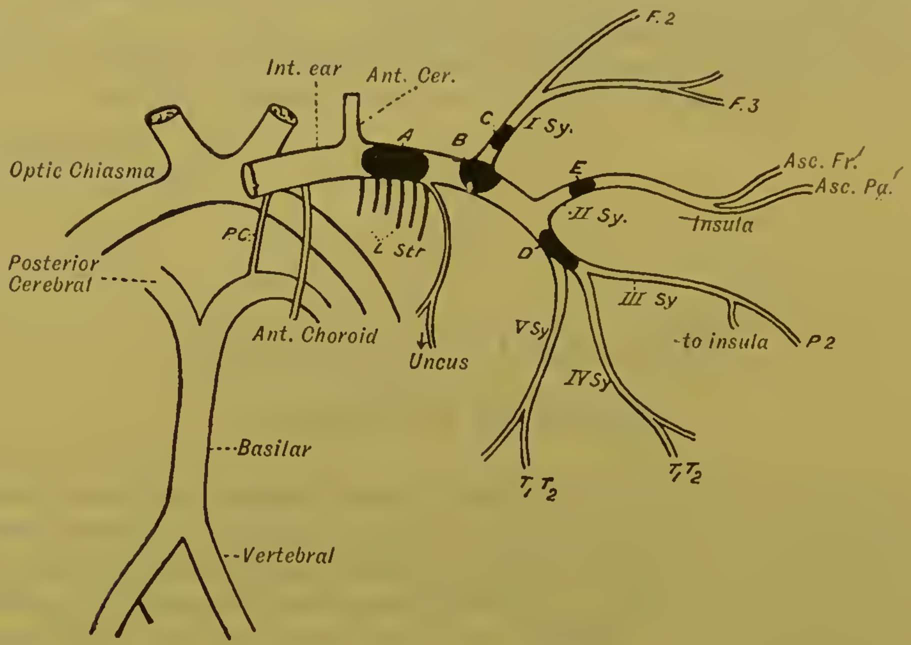

FIG. 13.—The diagram shows the arterial supply of the brain and the vessels entering into the formation of the circle of Willis *on one side*. The effects of occlusion of the middle cerebral artery in different situations is indicated. Occlusion at *A* will obstruct the artery at its commencement, and cause not only softening of the area of the cortex supplied by this vessel, but also of the basal ganglia and internal capsule by cutting off the blood supply to the lenticulo striate arteries. Obstruction at *B* will cause softening of the cortical area supplied by the middle cerebral. Each of the main branches may be obstructed at *C*, *D*, *E*, and the symptoms, especially if the lesion be on the left side, may be characteristic, *e.g.*, an obstruction at *C* might cause aphasia without hemiplegia; at *D* might cause visual and auditory word-blindness without paralysis; and at *E*, hemiplegia with aphasia; but the latter condition would not be so permanent as if the main artery were blocked, and there would be no word-blindness or word-deafness. (Modified after von Monakow.) The anterior communicating, however, is not shown.

ascending frontal, and ascending parietal, the island of Reil, the superior and inferior parietal lobule, and the first and part of the second temporal, including the tip of the temporal lobe and a portion of the under surface of the frontal lobe.

The anterior cerebral arteries supply the inner part of the under surface of the frontal lobe, the upper and anterior part of the frontal region, and the greater part of the mesial surface of the hemisphere, as far back as the internal perpendicular fissure.

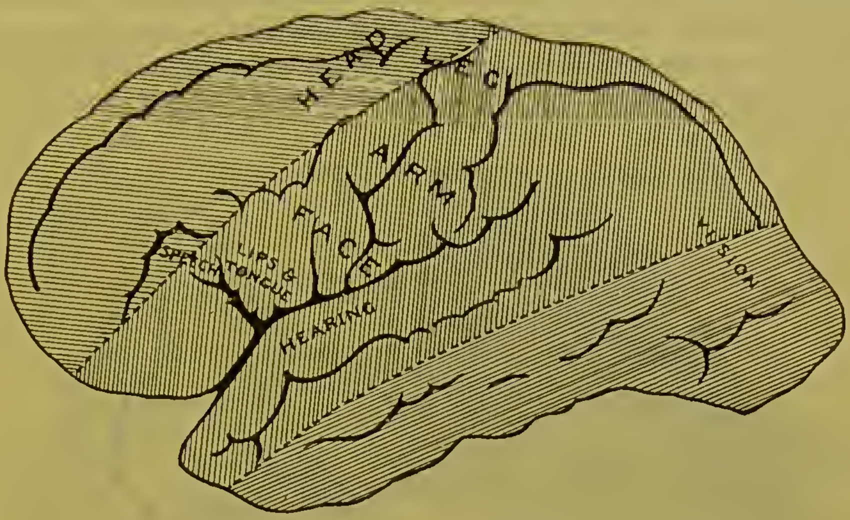

Fig. 14.—External surface of hemisphere to show diagrammatically by different shading the distribution of anterior, posterior, and middle cerebral arteries.

The posterior cerebral supplies especially the under surface of the temporal lobe and the occipital lobe. Both hæmorrhage and softening are more liable to occur in its area of distribution than in that of the anterior cerebral. The area of distribution of these arteries may vary slightly in different individuals, and a certain and variable amount of anastomosis may occur. Establishment of collateral circulation, when a cortical artery is blocked, is important in determining the size of the area

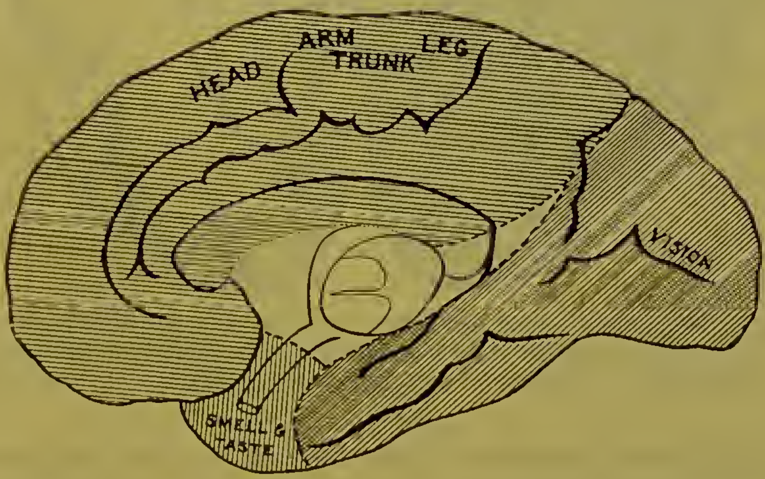

Fig. 15.—Mesial surface showing diagrammatically by different shading the distribution of the anterior, middle, and posterior cerebral arteries.

of softening, and therefore of the possibility of partial restoration of function; moreover, there may be some overlapping of arterial supply in some regions which possess special functions, *e.g.* blocking of the posterior cerebrals would certainly lead to softening of the occipital lobes and bilateral homonymous hemianopsy; but by collateral circulation through the middle cerebrals, a certain amount of central vision is still possible, because the cuneus, corresponding to the macular region, may still receive some blood from the middle cerebral; at any rate, this is the only explanation which can be offered to the cases of Förster and Schweigger. Again, it is said that the leg recovers before the arm in softening from thrombosis of the middle cerebral, because the anterior cerebral partly supplies the leg centre.

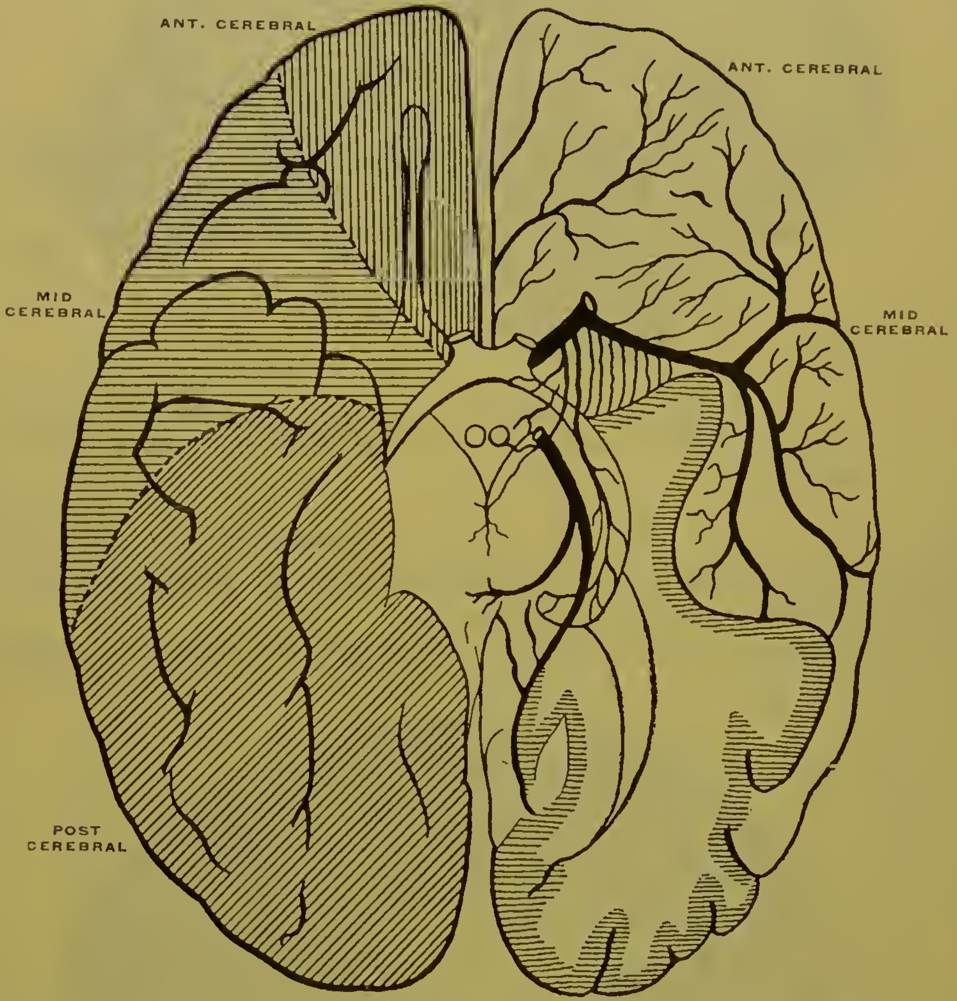

FIG. 16.—Diagram to show on the left side the arterial distribution of the base of the brain; on the right side the parts are partially cut away to show the anterior, middle, and posterior cerebral arteries and their branches.

Basal arteries.—The middle cerebral artery gives off two sets of branches, which penetrate the basal ganglia by the perforated space; they supply nearly the whole of the corpus striatum, as well as the internal capsule, and part of the optic thalamus. The smaller set, *internal*, supply the two inner portions of the lenticular nucleus and adjacent part of the internal capsule; the larger, *external*, are again divisible into an anterior set, *lenticulo-striate*, and a posterior, *lenticulo-optic;* the largest branch of the *lenticulo-striate* arteries is particularly liable to rupture, and the effused blood extends inwards, lacerating the fibres of the internal capsule (Fig. 17). It suffices to mention that the anterior cerebral arteries give off unimportant basal branches, and the posterior cerebral give off branches which are distributed to part of the optic thalamus, the crura cerebri, and corpora quadrigemina. These basal arteries supplying the basal ganglia, internal capsule, and adjacent structures are terminal arteries.

Basilar and vertebral distribution.—The cerebellum, like the cerebrum, is supplied by three arteries on each side—the inferior, middle, and superior; the two last named are always derived from the basilar; the inferior may, however, arise from the vertebrals. A large branch of the superior cerebellar which enters the grey nucleus, is the most liable to rupture in this situation, and the resulting hæmorrhage is generally large, and may occupy the whole of the corresponding lobe.

There is one important fact to remember about the supply of the pons and medulla, namely, that the delicate median arteries, which supply the nuclei of the important cranial nerves, are also terminal arteries.

Functions of the brain.—Cerebral localisation has been founded partly by experiments made upon the lower animals, either (1) by removing portions of the brain, and observing the loss of function, or (2) by electrical stimulation of the cortex and other excitable portions of the brain, or (3) by the association of clinical phenomena, observed during life, with lesions of the organ, observed post-mortem. Both the experimental and the clinical methods have controlled, corrected, and supported one another. We need only cite here the important clinical discovery of Hughlings Jackson, who showed that an irritative lesion of the cortex causes a discharge of the nerve cells, which proceeds in a definite march, causing a succession of representative movements in the head and eyes, face, arm, and leg of the opposite half of the body, as the excitation spreads. Fritsch and Hitzig first showed, experimentally upon animals, that the cortex is excitable by electrical stimulation. They further proved that when certain definite areas were stimulated, definite movements resulted; by carefully observing the area stimulated, and the resulting movement, they were enabled to map out, in the cortex of the dog, the excito-motor area, and thus to demonstrate and prove the existence of cerebral localisation. Ferrier first introduced the faradic method of stimulation, and mapped out the cortical centres in the monkey, and was the pioneer and founder in this country of very valuable experimental work on the subject of localisation. Munk first showed experimentally that removal of both occipital lobes caused blindness, and that removal of one caused blindness of the opposite half of the field of vision (hemianopsy). These results were confirmed by Schäfer in the monkey. Horsley and Beevor showed that the internal capsule could be mapped out into definite excito-motor areas, corresponding to the cortical areas, only proportionately less extensive. Numbers of other workers have added to our knowledge by experimental observations upon animals; but the reader is referred for further information to the "Text-Book of Physiology" (Schäfer). Of clinical workers the French school have added most to our knowledge in respect to cerebral localisation in man; in fact, it may be said to have had its foundation in the discovery in 1861 of the speech centre by Broca, after whom is named that portion of the brain corresponding to the third left frontal convolution, and its junction with the ascending frontal. Many important cases of localised patches of softening have been associated with loss of function observed during life. Various monoplegias have been noted, by which the Rolandic area has been mapped out into definite regions, closely corresponding to those mapped out by stimulation in the orang utang by Horsley, who has, moreover, for diagnostic purposes, stimulated the cerebral cortex in man, and shown that the facts taught by experiments upon animals are correct (Fig. 17). There are, however, functions of the human brain which can only be ascertained by association of defects observed during life with lesions post-mortem, *e.g.* the centres connected with (1) articulate speech, absence of which is termed motor aphasia, localised in Broca's convolution; (2) visual word memory, localised in the angular gyrus; (3) auditory word memory—thus cases of softening of the posterior third of the first temporal convolution have been observed in persons who could read written language, but could not understand spoken language; (4) a few cases of disease of the base of the second frontal convolution have been found associated with inability to write words, but not inability of utterance. For further particulars, *vide* article "Aphasia."

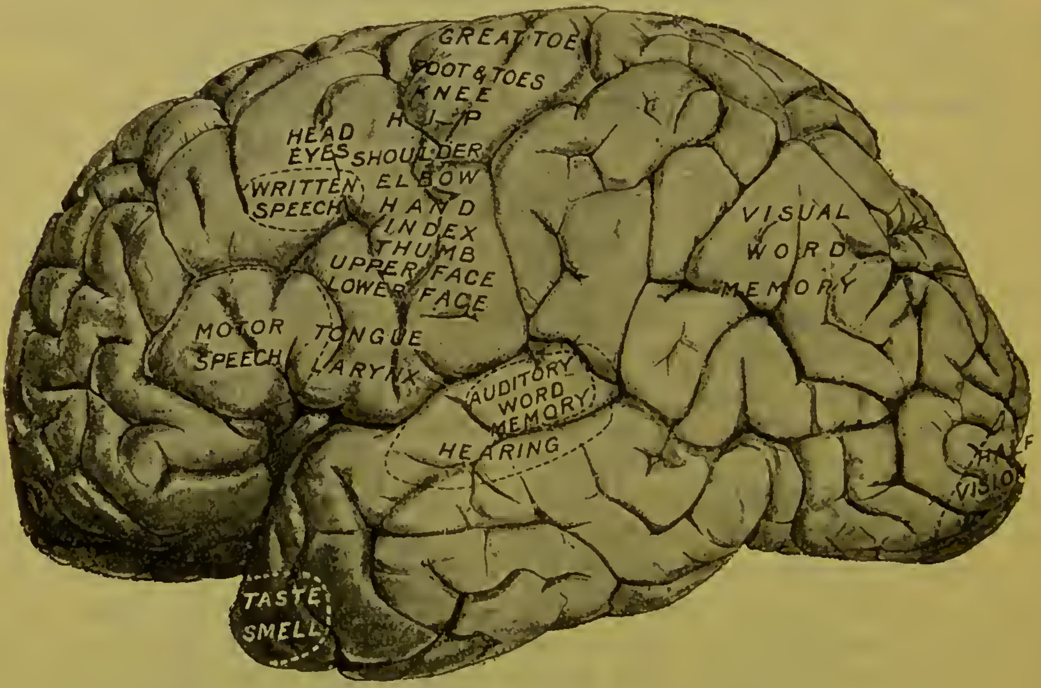

FIG. 17.—Diagram illustrating the probable situation of the cortical centres in the left hemisphere of man. It has been composed partly by the acceptance of the records of stimulation experiments in the orang utang and man, and partly from the association of defects observed during life with lesions found post-mortem.

According to Flechsig, certain areas of the cortex contain only neurons of association and no neurons of afferent or efferent projection; these latter are found, according to him, only in the excito-motor (Rolandic) region of the cortex and those regions which are concerned with special sense, *e.g.* occipital lobes, first temporal and tip of the temporo-sphenoidal lobe, which are the convolutions bounding the primary fissures, Rolandic, sylvian, calcarine; all the remainder of the cortex consists of association centres. It is probable, both from the effects of disease, as well as of extensive injuries, that the frontal lobes are concerned with the higher functions of mind, as impairment of the moral and emotional faculties has been the only result of extensive destruction of the cortex in this region. We have shown that there is abundant evidence to prove that the departure platform of the efferent motor projection system is in the central convolutions (Fig. 18). The neurons of this system are physiologically connected by their dendrons with the terminals of the afferent projection system, and by the tangential system of fibres of the superficial layers of neurons, by which co-ordinate action of adjacent systems is maintained. In every voluntary movement the whole three nervous circles (cerebral, cerebellar, and spinal) are in action; impulses are ascending the afferent systems and descending the efferent during the whole time. We are conscious of the position of our limbs by the sensations which ascend the afferent system, and this consciousness is necessary to and precedes volitional movement; the sense of movement (kinæsthesia, Bastian) is a combination of the sensations proceeding from skin, muscle, tendons, and joints; *a priori* we should expect the arrival platform of these sensations in the cortex to be in close proximity to the departure platform of the efferent system. Flechsig, by the embryological method, has shown that the sensory fibres of the internal capsule terminate in the central convolutions. It is true that lesions of the cortex do not produce marked sensory defects as a rule, unless there is a very large region involved; however, I have had the opportunity of testing patients upon whom Horsley had operated for tumour, and some of those undoubtedly showed marked sensory defect; moreover, the sensory aura that frequently precedes epileptiform convulsions in Jacksonian epilepsy, serves to show that the central convulutions of the cortex are sensori-motor.

Regional diagnosis.—Bearing these facts concerning cortical localisation in mind, a regional diagnosis of a cortical lesion can be made, if it be situated in a region which has a definite function. If it be an irritative lesion, and situated in the excito-motor area, it will cause fits, commencing usually with a sensory aura in the part which is thrown into spasm. If it be a destructive lesion, there will be a loss of function which may or may not be discoverable, according to the side of the brain affected, and its seat. Loss of speech, of visual word-memory, of auditory word-memory, or agraphia, only occur when the centres in the left hemisphere are destroyed, unless the patient be a born left-handed person. Extensive lesions in some portions of the brain, *e.g.* frontal region, may not be discoverable during life, but probably this is owing to our own want of discernment and of previous knowledge of the intellectual and moral character of the individual before he was afflicted.

Reference to Fig. 13 will help the reader to understand the effect of occlusion of the middle cerebral artery and its branches. If the artery be blocked at its commencement, there will not only be softening of the whole cortical area supplied by this vessel, but also of the internal capsule and basal ganglia, resulting in hemiplegia; and if on the left side, there will be aphasia, word-deafness, and word-blindness. If the first Sylvian branch is blocked, there will be softening in Broca's convolution and aphasia. If the second, there will be softening of the Rolandic area and hemiplegia; while blocking of the posterior main division of the middle cerebral on the left side will cause mainly word-blindness and word-deafness, but not aphasia or hemiplegia. The effect of occlusion of the posterior cerebral artery is lateral homonymous hemianopsy, often only partial in character.

Centrum ovale.—Lesions in the centrum ovale produce effects according to the size and seat of the lesion. The motor and sensory projection systems form two funnels, with their base at the cortex and their neck at the internal capsule. It requires a large lesion in this region to interrupt

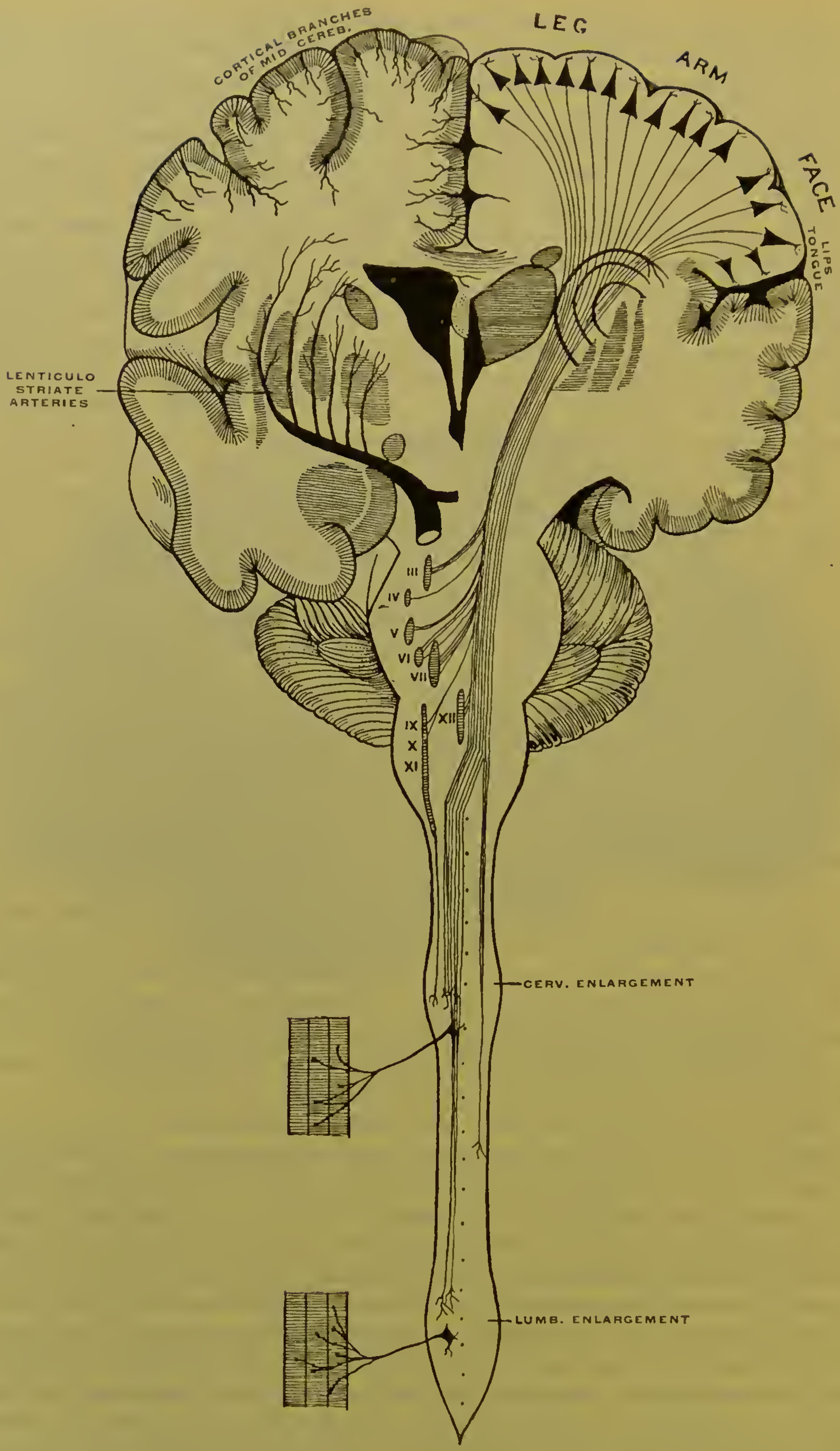

FIG. 18.—Diagram to illustrate the cortical pyramidal system of neurons. On the left side is seen the cortical branches of the middle cerebral artery, obstruction of which would cause softening of the sensori motor area of the cortex, degeneration of the cortico-pyramidal neurons, and subsequently secondary lateral sclerosis. The lenticulo striate arteries

the whole of the fibres belonging to these systems. The writer has seen multiple softening in this region produce a hemiplegia, in which a certain amount of recovery took place, but was afterwards followed by epileptiform seizures on the partially paralysed side, the patient dying from thrombosis of the basilar. He has also seen a case of hæmorrhage in this region which during life simulated softening, for it was attended by only partial loss of consciousness and epileptiform convulsions in the paralysed limbs; it terminated fatally by effusion of blood into the lateral ventricles. The result, then, of a lesion in this situation may be irritative epileptiform convulsions and contracture, or paralysis with hemiplegia or monoplegia, and sometimes hemianæsthesia. A lesion in the posterior part of the hemisphere might cause a hemianopsy by destruction of the optic radiations to the occipital lobe. If the lesion is in the left hemisphere, however, the paralysis may be associated with some form of aphasia.

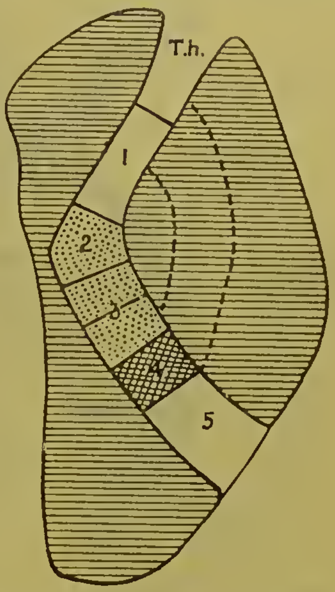

Fig. 19.—Scheme of the internal capsule, after Obersteiner. To the right is the lenticular nucleus. To the left in front is the nucleus caudatus, and at the back the optic thalamus. T.h. Cortico thalamic fibres; 1, frontal fibres to the pons; 2, cortical efferent system to motor cranial nuclei; 3, cortical pyramidal system to motor nuclei of cord; 4, muscular sense; 5, sensory crossway.

Internal capsule. — Both softening and hæmorrhage in this region is very common. Reference to the diagram (Fig. 19) shows that both the motor and sensory projection systems of fibres are liable to destruction, and if the lesion is extensive, so as to involve the whole posterior half, hemiplegia and hemianæsthesia may ensue. Sometimes the leg is more affected than the arm and face; from the situation of the fibres it will be seen that in such a case hemianæsthesia is much more likely to occur; whereas if the face is affected, and the arm only slightly, it points to a small lesion in the anterior third of the posterior half. The lesion most frequently affects the anterior two-thirds of the posterior half, and causes hemiplegia of the opposite side. Should the lesion be on the left side, there is temporary aphasia associated with paralysis.

Lesions of the basal ganglia.—There is no essential difference in the symptoms which have been observed in cases of softening of the striate from those of the lenticular nucleus. Charcot has pointed out that frequently there is a transitory and ill-defined paralysis. The effects of lesions in these situations thus contrast markedly with those of the internal capsule, where the paralysis is usually well defined, persistent, and apt to be followed by rigidity. The writer has recently seen such a case in a woman who had both upper and lower limbs paresed, the weakness being more marked on the right than on the left side. The patient seldom

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are also shown, the longest and most external of which is especially liable to undergo rupture and produce apoplexy. On the right side the cortico-pyramidal neurons are represented with their axis cylinders proceeding to the motor nuclei in the crus, pons, medulla, and spinal cord, decussation occurring in the great majority. In the capsule is represented the progressive destructive effects of a hæmorrhage in cutting off the fibres in its different parts.—Modified from van Gehuchten.

spoke, and frequently burst into tears. There was found post-mortem softening in both lenticular nuclei, the left being more affected than the right. No certainty of diagnosis exists when there is a lesion of the optic thalamus; like the corpus striatum, the effects are due more to injury of adjacent structures in the internal capsule and the optic tract; consequently, hemianæsthesia and hemianopsy may occur in lesions of the thalamus.

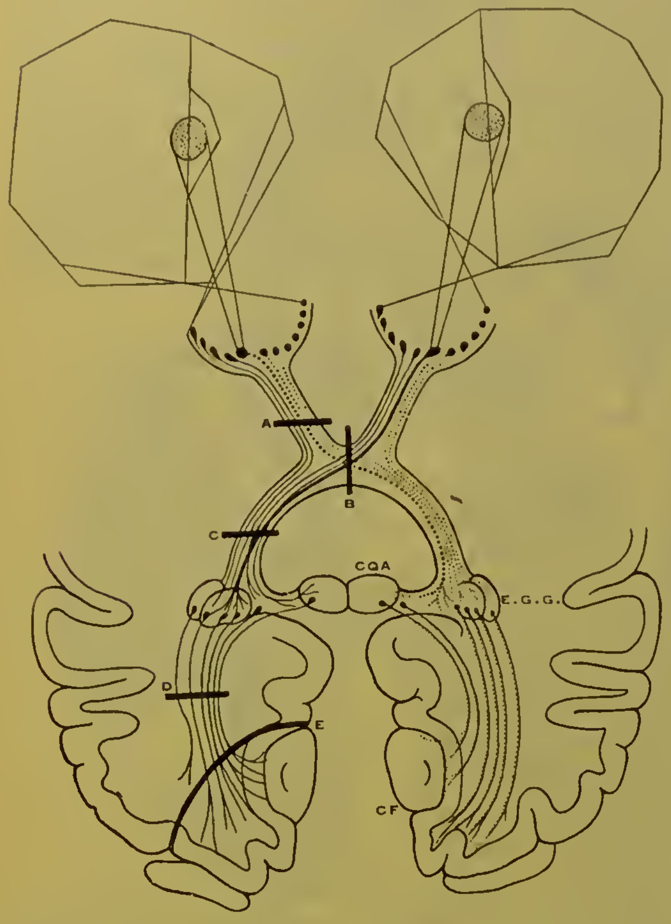

FIG. 20.—Diagram to illustrate lesions of the visual path. A lesion at *A* will produce unilateral blindness. A lesion at *B* is very liable to produce bitemporal hemianopsy by destroying the fibres coming from the two inner halves of the retina. A lesion at *C*, involving the optic tract on the left side, would cut off the outer half of the field of vision of the right side, and the inner half of the field of vision of the left side; that is right lateral homonymous hemianopsy. Lesions at *D* and *E* would also produce this phenomenon, but these lesions being situated behind the corpora quad. *C*, *Q*, *A*, to which fibres concerned in the light pupil reflex are given off, would be distinguished from lesions at *C* by the presence of Wernicke's pupil phenomenon. The dotted circle in the field of vision on either side represents the fixation point. The ganglion cells of the retina are represented by black dots and ovals, from which dotted and fine black lines respectively proceed to represent the nerve fibres of first neurons of the visual path; these terminate in the external geniculate bodies and the corpora quadrigemina, from the former the visual path is continued by another set of neurons, whose terminal arborisations are in the half vision centre of the occipital cortex of the same side. The area of the macula lutea which is concerned with distinct vision is represented by blacker dots and lines in the visual path.—Modified after von Monakow.

Lateral ventricles.—In cerebral hæmorrhage from rupture of a miliary aneurysm on the lenticulo-striate artery, frequently the effused blood tears through the brain substance and enters the lateral ventricles. In a case of apoplexy with hemiplegia, it may rupture primarily into the ventricle, or there may be secondary hæmorrhage and rupture into the ventricle, with accession of new and fatal symptoms. The condition it may be mistaken for, is hæmorrhage into the pons. Conjugate deviation towards the lesion and away from the paralysed side, with some slight evidence of sensibility (slight conjunctival reflex) on the side to which the eyes are turned, if the coma be not too deep, associated with unilateral convulsions of the paralysed limbs and unequal pupils, would point to one lateral ventricle being filled. Sometimes, however, the blood effused is sufficient to fill both lateral ventricles and the third and fourth ventricles, and, passing thence beneath the arachnoid, it surrounds the medulla and pons. The pupils in such a case may be contracted and insensitive, and the paralysis being bilateral, it is easily understood that this condition may be mistaken for a pontine hæmorrhage.

Visual paths and centres.—Unilateral blindness may be the result of optic atrophy. A lesion at the chiasma, *e.g.* tumour of the pituitary body, acromegaly, or fracture of the base of the skull, may injure the optic chiasma and destroy the path from each nasal half of the retina, consequently there would be loss of vision of both sides (bitemporal hemianopsy) (Fig. 20). If the lesion involves the optic tract in the external geniculate body, or the optic radiations or the occipital lobe, the most common form of hemianopic defect occurs, namely, homonymous lateral hemianopia; *e.g.* lesions at *CDE* would cause blindness to the right of the mid-line, because the path from the temporal field of the left and the nasal field of the right eye would be interrupted. Incomplete forms of lateral hemianopsy generally indicate softening or disease in the occipital or suboccipital cortex.

Tumours and lesions of the corpora quadrigemina may be associated with blindness and abolition of the pupillary reflex; the nuclei of the third nerve may be involved, and there may be bilateral paresis or paralysis of the third nerve in association with disorders of equilibration and locomotion. When quadrigeminal disease is suspected, Wernicke's hemiopic pupillary reaction should be tried. To do this, a fine pencil of light should be thrown on the sensitive half of the retina; contraction of the pupil promptly results; but there is no response when thrown on the insensitive half. Absence of this symptom shows the lesion behind the geniculate body, either in the optic radiations or the occipital cortex. Oppenheim's method of testing incomplete hemianopsy is useful, especially the binasal or bitemporal forms. Two similar objects are presented to the patient at the same time, and in such a way, that one lies in the outer and the other in the inner half of the field of vision. If the patient be now asked what he sees, he will point to one object in the clear half of the field of vision, and neglect the other.

Cerebellum.—Luciani has shown that ablation of this organ in animals produces asthenia, atonia, and astasia. Russell has confirmed Luciani's results. It is generally admitted that the cerebellum is an organ concerned with muscular co-ordination, and it is probably by the exercise of this function, under the guidance of peripheral stimulation, that it serves to maintain steadiness in gait and station. Each lateral lobe of the cerebellum is connected with the motor cortex of the opposite cerebral hemisphere. Probably the cerebellum has also an important influence upon the maintenance of tonus of correlative antagonistic muscles in the fixation of a joint.

The most important clinical phenomena which have been observed in cerebellar disease are unsteadiness of gait and station, paresis or paralysis on the same side as the lesion, sometimes on the opposite side, due in all probability to pressure upon the pyramidal tract before decussation. Ataxy is the most important diagnostic symptom, the gait is peculiar and oscillating, the patient swaying to and fro like a drunken man. There may be a tendency to fall forward or backward or towards one side, and it is generally a destructive and not an irritative lesion that exists. As a rule the patient walks and tends to fall towards the affected side in unilateral lesions. There is generally hemiparesis on the same side as the lesion. I saw a case recently in which the side was correctly diagnosed and successfully operated upon by trephining over the lateral lobe on the hemiparetic side. Vertigo is a very constant symptom, and it is noteworthy that it occurs more frequently with cerebellar affections than with lesions in any other region of the brain; it is probable that this is due to the destruction of the receptive mechanism of impressions from the semicircular canals. Hydrocephalus frequently occurs in children, when there is a tumour of the cerebellum due to obstruction of the veins of Galen. Stupor is a common symptom, likewise vomiting and severe optic neuritis are generally present in tumour or abscess of the cerebellum. Adjacent structures, *e.g.* pons, crus cerebri, corpora quadrigemina, medulla oblongata, or cranial nerves, may be pressed upon by a large tumour, hæmorrhage, or abscess, and other symptoms may therefore exist, rendering the diagnosis more difficult.

Crus cerebri.—A lesion of the crus cerebri usually produces hemiplegia of the opposite side of the body, but paralysis of the motor oculi of the same side; this is termed the syndroma of Weber. The face will be drawn towards the side upon which there is ptosis and external strabismus, the tongue deviates to the paralysed side of the face and limbs. Sensibility may or may not be involved, according to the situation and extent of the lesion; if it involves the outer part (Fig. 21), there will be interruption of the sensory tract and hemianæsthesia. The symptoms would then be very like those produced by a lesion of the hinder part of the posterior limb of the internal capsule.

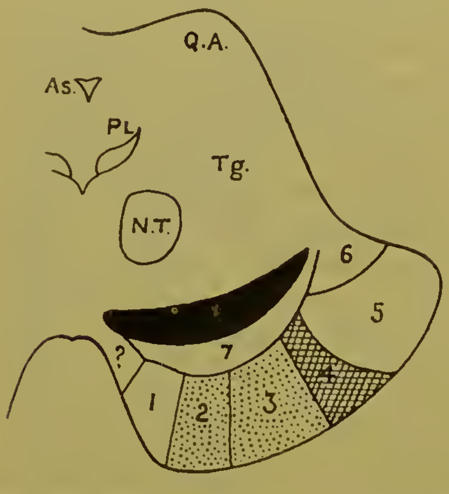

Fig. 21.—Diagram of the cerebral peduncle. Q.A. Anterior corpus quadrigeminum. As. Aqueduct of Sylvius. N.T. Red nucleus. Tg. Tegmentum. P.L. Posterior longitudinal bundle. 1. Frontal pontine tract. 2. Tract to motor cranial nerves. 3. Pyramidal tract. 4. Muscular sense tract. 5. Sensory fibres of the crus. 6. Fillet fibres. 7. Stratum intermedium.—Obersteiner.

Pons varolii.—Lesions in the central regions of the pons, according to Bastian, "are characterised by irregular bilateral motor paralysis of the face and limbs, well-marked diminution or alteration of sensibility in some parts of the body, together with some distinct difficulty in deglutition as well as articulation."

The *upper lateral region of the pons.*—Hemiplegia is a result on the side opposite the lesion; for the pyramidal system of fibres from the cortex have not decussated, nor is the facial nucleus or nerve involved. The fifth nerve may, however, be implicated on the same side as the lesion, especially its motor root, with a consequent weakening of the muscles of mastication on the side of the lesion.

The lower lateral region of the pons.—Alternate hemiplegia or cross paralysis is the characteristic condition met with in this situation. There is unusually well-marked facial paralysis on the side of the brain lesion, and hemiplegia of the opposite side. Since the facial nucleus or nerve is injured, the paralysis may affect the upper as well as the lower facial muscles, and the muscles may undergo more or less degeneration, as in a peripheral facial paralysis. There are certain conditions frequently met with in any of the above-mentioned pontine lesions, namely, emotional weakness, an undue tendency to burst into tears or laughter; polyuria, and conjugate deviation of the head and eyes.

It may be opportune here to explain this important symptom, which we have seen may be met with in various lesions, and which, when present, forms a valuable aid in diagnosis. Conjugate deviation of the head and eyes depends upon the associated action of certain muscles of one side with certain other muscles of the opposite side. Thus the external rectus of one side acts with the internal rectus of the other, and to produce the lateral movement of the eyes, *e.g.* to the left, we must have associated contraction of the left external rectus with the right internal rectus; the antagonists would be the right external rectus and the left internal rectus. It is obvious that there is always a correlative antagonism between the two groups of muscles which move the eyes laterally; consequently, either paralysis or over-action of one group will lead to deviation.

Now the head is moved laterally by the contraction of two different groups of muscles, the splenius capitis, the recti capitis posteriosis, and the great oblique muscles of the same side, acting in association with the sterno-mastoid and trapezius of the opposite.

Thus the muscles engaged in conjugate deviation to the left and right respectively would be—

RIGHT.		LEFT.
Right external rectus Left internal rectus	→ ←	Left external rectus—sixth nerve. Right internal rectus—third nerve.
Right splenius capitis, recti capitis, and great oblique Left sterno-mastoid and trapezius	→ ←	Left splenius capitis, recti capitis, and great oblique—cervical nerves. Right sterno-mastoid and trapezius—spinal accessory.

Since there is correlative antagonism between the two groups, over-action on the part of one group or paralysis of the other will cause conjugate deviation of the head and eyes. This associated movement has a definite local representation in the cortex; stimulation of a region in the orang utang, corresponding to the base of the second frontal in man, causes lateral deviation of the head and eyes away from the side which is excited; stimulation of the central portion of the mesial surface of the occipital lobe, corresponding to the macular region, also produces lateral deviation of the eyes to the opposite side. It was shown by the writer and Schäfer that simultaneous stimulation of the two frontal or two occipital areas, with currents of equable intensity, produced a balance in the correlative antagonism of the groups of muscles concerned in lateral deviation of the head and eyes of the two sides; the result being that the animal looked straight forward, as in concentrated attention. Unequal strength of the stimulus, or varying degrees of excitability of the cortex, invariably produced a lateral deviation.

Reference to the diagram will show how these facts may be applied clinically. A large hæmorrhage destroying the corona radiata, or even a small one, provided it lacerated the knee of the internal capsule (Fig. 22), would cut off the impulses coming from the cortical regions, which represent the excito-motor area. The result is a loss of balance in the tonus of the two groups of correlative antagonistic muscles, and conjugate deviation of the head and eyes; *e.g.* if the lesion be (as represented in the diagram) in the left hemisphere, the head and eyes will be turned to the left. The patient then persistently looks, in a paralytic lesion, towards the seat of the injury, and away from the paralysed face and limbs. As Prevost, who first gave a complete description of this important localising symptom, showed, if the patient's head be forcibly turned towards the mid-line, it will not remain there, but the deviation is resumed upon the operator taking his hands away from the head. If the lesion is in the lower part of the pons, both the facial nucleus and the sixth nucleus may be involved; the result will be paralysis of the right half of the face, and the left arm and leg; with this alternate paralysis there may be conjugate deviation of the head and eyes to the left, owing to over-action of the left external rectus and right internal rectus, which are unopposed by their paralysed antagonists. The associated deviation of the head is no doubt due to the muscles of the sixth being the principal nervous structure concerned with this associated movement, and it is probable that fibres from it pass to the third nucleus of the opposite side, as figured in the diagram (Fig. 22), by means of the posterior longitudinal bundle, and also downwards by this tract to the spinal accessory nucleus of the opposite side, and the anterior horn cells of the cervical nerves of the same side. This is not figured in the diagram, to avoid complication. The patient would therefore, in a lesion situated in the lower half of the right side of the pons, look away from the lesion and the paralysed half of the face, but towards the paralysed arm and leg, the pyramidal fibres of which have not yet decussated. Conjugate deviation usually passes away in the course of a few days.

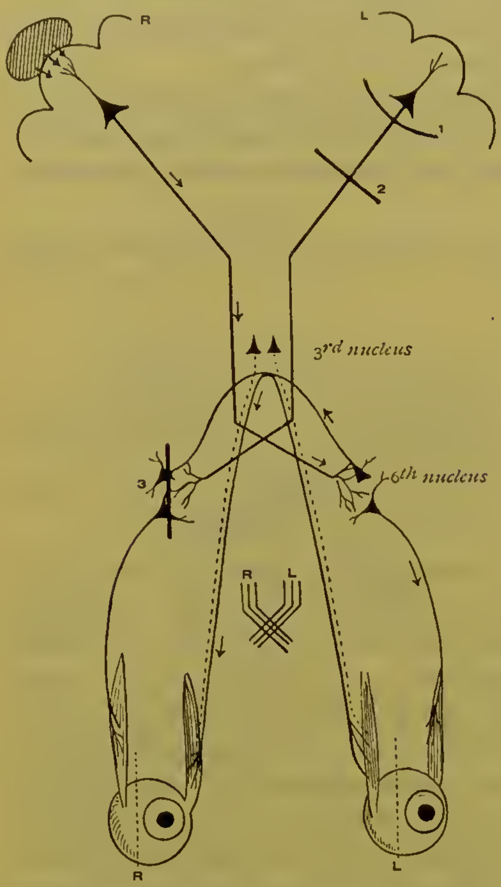

Fig. 22.—Diagram to represent the mechanism of conjugate deviation. R. Right hemisphere with a tumour on the surface producing cortical irritation and discharge. L. Left hemisphere with destructive paralytic lesions at 1 and 2. At 3 is represented a destructive lesion in the pons, involving the sixth nucleus on the right side. All these conditions will produce conjugate deviation of the head and eyes to the left. Reference to this diagram will enable the reader to understand the description of this phenomenon in the text.

Hitherto we have only referred to paralytic lesions; irritative lesions may occur,—*e.g.* tumours, meningeal hæmorrhages, cysts, etc., seated in certain regions of the cortex, might cause an increased cortical discharge and Jacksonian epilepsy. Clonic spasms occur in the group of muscles which move the head and eyes, *e.g.* to the left, as shown in the schema; the balance in correlative antagonism is overturned, and with each cortical discharge conjugate deviation takes place. The patient then looks away from the lesion, and towards the side upon which clonic spasms in the face and limbs are occurring. If there is an irritative lesion in the pons, the patient looks towards his lesion. According to Grasset, paralytic or irritative lesions of the cortex, producing conjugate deviation, have been found most frequently situated about the bottom of the fissure of Sylvius and the angular gyrus.

Medulla oblongata.—Lesions in this situation may present symptoms closely resembling those met with in the pons, *e.g.* bilateral paralysis more or less complete occurs; but a consideration of the results of injury to the lower, as distinguished from the upper, cranial nerve nuclei, will help in a correct diagnosis. Thus a bulbar lesion would be indicated if there is marked difficulty of articulation and deglutition, if the facial paralysis is incomplete and bilateral, or the orbicularis oris is affected while the upper part of the face escapes. There is frequently loss of phonation with paralysis of the tongue and palate and larynx, and loss of reflex excitability of the pharynx, Cheyne-Stokes respiration, glycosuria, polyuria, or albuminuria, and slow, irregular, or rapid action of the heart.

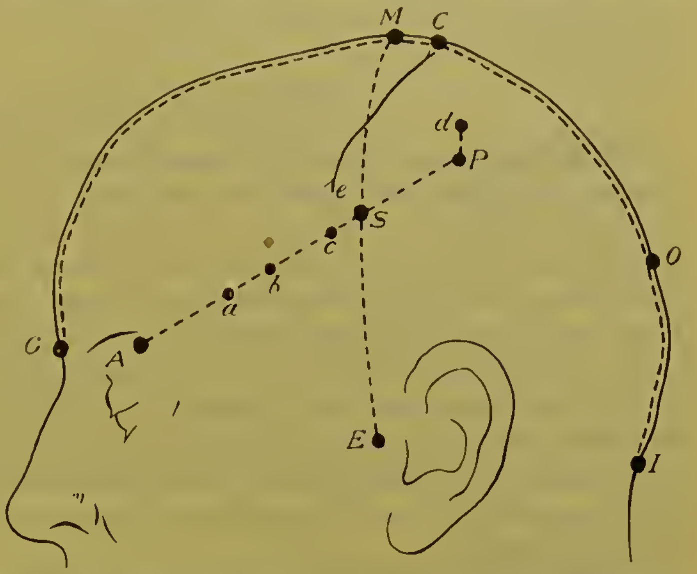

Fig. 23.—Cranio-cerebral guiding lines (after Anderson and Makins) traced upon a photograph of one of the casts prepared by Professor Cunningham. The figure and description (abridged) is taken from Mills. (1) A median line *sagittal* is drawn from *G*, the glabella, to *I*, the external occipital protuberance (inion). (2) A line *frontal* from the mid-sagittal point (*M*) to the depression just in front of the ear at the level of the upper border of the meatus (*E*). (3) A line squamosal from the most external point of the external angular process at the level of the superior border of the orbit (*A*), to the junction of the middle and lower thirds of the frontal line (*S*), and prolonged for about 1½ in. behind the frontal line to the parietal point (*P*). The upper extremity of the central Rolandic fissure (*C*) lies between the mid-sagittal point (*M*) and a point ¾ in. behind it, and the lower extremity of this fissure (*e*) is situated about ¾ in. in front of the junction of the squamosal and frontal lines. The commencement of the Sylvian fissure (*a*) is situated about 1½ to 2 in. behind the external angular process along the squamosal line, the bifurcation of the fissure (*b*) of Sylvius is seven-twelfths of the distance from *A* to *S*. The termination of the fissure of Sylvius (*d*) is ½ in. above *P*, in a direction parallel to frontal line. The parieto-occipital fissure *O* is situated on the sagittal line seven-twelfths of the distance from *M* to *I*.

Cranio-cerebral topography.—It is of practical importance to know the relation of certain portions of the brain to definite points and sutures of the skull. The accompanying diagram, by Anderson and Makins, after Cunningham, will give the reader a good general idea of the relation of the brain to the skull, but it is not within the scope of this article to give a full description of cranio-cerebral determinations, and the various methods which have been adopted by surgeons for localising the situation through the skull of the various fissures and convolutions, for the purpose of assisting him in trephining successfully for such affections as tumour, abscess, intra-cranial hæmorrhage, fracture of the inner table, mastoid abscess, disease of the Gasserian ganglion, meningitis, and other affections (Fig. 23).

The approximate situation of the various motor centres can be determined by many methods. It is, however, recognised by surgeons that all fixed methods are only approximate, for the situation varies with the shape of the skull in different individuals.

F. W. MOTT.

PACHYMENINGITIS.

By this term an inflammation of the dura mater is meant, and a distinction between an external and internal variety has been drawn, according to whether the inflammation first affects the outer or inner layer of the membrane.

External Pachymeningitis.

Etiology and morbid anatomy.—The external variety is nearly always secondary to injury of the bones of the skull, or to caries or necrosis of them, due either to extension from middle ear disease, etc., or to syphilis. Rarely an inflammatory process like erysipelas of the scalp gives rise to it, while very rarely no cause can be found. In the earlier stages the dura is œdematous and congested, while later it is covered by pus, which separates it from the bone, and also infiltrates its substance. Where there has been no formation of pus, the thickened outer layer of the dura mater becomes firmly adherent to the bone of the skull. On the other hand, the inflammation may spread through the whole thickness of the membrane, and the pia-arachnoid may thus become united to the dura by lymph.

Symptoms.—The symptoms are indefinite; but there may be rise of temperature, headache, and delirium, and convulsions may occur, or motor paralysis corresponding to some part of the cortex pressed on by a collection of pus.

Treatment.—This must be directed to the cause, whether injury, disease of bone, syphilis, or inflammatory conditions outside the skull. If there is evidence of the formation of pus, the skull should be trephined to allow of its escape. In other respects the treatment does not differ from that of other forms of meningitis to be subsequently described.

Internal Pachymeningitis.

This is either hæmorrhagic, constituting hæmatoma of the dura mater, to be presently described; purulent, in which case it is almost always associated with a similar inflammation of the pia-arachnoid, or of its own outer layer, from both of which conditions it is indistinguishable clinically; or syphilitic. Pachymeningitis hæmorrhagica is also known as hæmatoma of the dura mater, and consists in an effusion of blood on the inner surface of the dura, occupying a considerable area over one or both cerebral hemispheres.

Etiology.—The condition is met with in those over 50 years of age, and males suffer more frequently than females. The subjects of chronic mental diseases are peculiarly liable to be affected, especially general paralytics and chronic alcoholics. It also occurs in persons with chronic renal and cardiac disease with degenerate cerebral vessels. Diseases in which the hæmorrhagic diathesis is marked, as in profound anæmias, scurvy, and scorbutic rickets, are sometimes responsible for its occurrence, as are acute diseases, such as enteric fever, smallpox, typhus, relapsing fever, and acute rheumatism. An injury to the head has also been followed by this condition.

Morbid anatomy.—The exact condition met with varies in different cases. There may be only a delicate membrane, beneath the dura and easily separable from it, consisting of interstitial tissue and numerous capillaries, and dotted with minute hæmorrhages; or the membrane may be formed by a series of laminated clots, 3 to 5 mm. in thickness, the more recent parts of which are soft and red, owing to small vessels and extravasated blood, while the older parts are firmer and paler owing to the formation of fibrous tissue. A series of cysts may be met with in the meshes of the membrane, and may contain liquid or coagulated blood variously altered. The condition is bilateral in about half the cases, and is usually associated with atrophy of the convolutions, while in some instances the extravasation is so large that marked compression of one or both cerebral hemispheres results, a large space being thus created between the dura and the surface of the hemisphere.

The false membrane may be adherent to the dura alone or to the arachnoid as well, while in some cases, in which no such adhesions to the dura exist, it may be difficult to disentangle it from the arachnoid. The dura itself, when separated from the false membrane, may show no thickening or other abnormality, and its under surface may be quite smooth. In eight cases which Bondurant examined he found inflammation of the arachnoid in all, while inflammation of the dura was either absent or insignificant. He contends that the term chronic hæmorrhagic leptomeningitis would be more appropriate than that by which the condition is generally known. Cases which I have examined lead me to conclude that the condition is primarily an affection of the dura, and that the blood is derived from the vessels of this membrane, though Huguenin holds that it is derived from the vessels of the pia. With a source of irritation in such close proximity, it is not surprising that the pia-arachnoid should become secondarily affected.

Pathology.—Virchow holds that the condition is primarily inflammatory, a delicate vascular membrane being formed before there is any hæmorrhage into it; while others, like Huguenin and Wigglesworth, regard the hæmorrhage as the primary lesion. The question is far from settled, but the post-mortem evidence at our disposal suggests that both forms of origin occur; and it is difficult to see how it could be otherwise, if we consider how varied the etiological conditions are under which the affection occurs. Some of these make the inflammatory theory highly probable, while others not only make it improbable, but support strongly the view that hæmorrhage is the primary lesion.

Symptoms.—The symptoms are most indefinite. A considerable proportion of cases of this affection are met with post-mortem, in which there have been no symptoms to cause it to be suspected during life; other cases probably give rise to symptoms, but these are masked by those consequent on the condition to which the hæmatoma is secondary; while in a third class of cases the symptoms which occur simulate those due to other cerebral affections, for which this condition is accordingly mistaken. Such symptoms as result vary according to the positions of the lesions and their sizes. As a rule the onset is sudden, and apoplectiform seizures recur from time to time as fresh hæmorrhages take place; the patient may recover apparently completely from one attack, when fresh symptoms arise in consequence of new hæmorrhages; and it is this repetition of attacks of cerebral disturbance that is so characteristic of hæmatoma of the dura mater. The general symptoms of compression consist in headache, vomiting, slow irregular pulse, general muscular weakness, contracted pupils, somnolence, and coma. Nystagmus may occur, and optic neuritis is present now and then towards the end of life. If the hæmatoma is situated over the convolutions bordering on the fissure of Rolando, it may give rise to unilateral convulsions or paralysis, or to similar conditions affecting one limb chiefly, or one side of the face. Aphasia is not uncommonly met with when the left hemisphere is affected.

In the most severe cases death may result from the first attack of hæmorrhage which gives rise to symptoms, while more commonly there are alternations of improvements and relapses, which may spread over months or even years, until the condition is ultimately fatal. In the only properly authenticated case of recovery from this affection, the patient was murdered six months after all symptoms had disappeared, and the remains of the morbid process were found on post-mortem examination.

In children the disease cannot be recognised with certainty, but cerebral symptoms occurring in the course of scurvy rickets may lead to hæmatoma being suspected. The same may be said with regard to the occurrence of such symptoms after, or in association with, any of the known causes of the affection.

Diagnosis and prognosis.—While cerebral symptoms, such as those that have been detailed above, occurring in association with one of the known causes of the condition, may lead to its being suspected, there is nothing distinctive about the symptoms which justify an absolute diagnosis, as a rule. A sudden onset, with cortical symptoms, either convulsive or paralytic, and a course in which there are alternations of improvements and aggravations, may, however, lead to its recognition. When pronounced enough to give rise to symptoms by which it can be recognised clinically, it is almost certain to be fatal; whereas in slighter cases, in which the condition has not been suspected during life, but has been found post-mortem, there is often nothing to suggest that the hæmatoma in itself would have been fatal, had its cause not been attended with this result.

Treatment.—If recognised or suspected, the patient should be kept at rest in a quiet, well-ventilated room, with the head and shoulders raised, care being taken to avoid flexion of the neck with consequent obstruction to venous return, and cold should be applied to the head. Stimulants and cardiac tonics must be avoided as far as possible, care must be taken not to allow the volume of the circulation to be increased by the drinking of too much liquid, and, if necessary, means must be taken to reduce arterial pressure by depletion, and in any case the bowels must be kept freely open. When possible the cause should be removed or counteracted; and if localising signs can be detected, pointing to the presence of a large extravasation in an accessible position, the case should be submitted to the surgeon. The possibility of any of these measures being successful is, however, very slight.

J. RISIEN RUSSELL.

LEPTOMENINGITIS.

In this form of meningitis the pia and arachnoid are affected, and the exudation takes places between the two membranes.

Etiology.—In addition to the form of this affection due to the presence of tubercles in the membranes, and that due to syphilis, the variety known as epidemic cerebro-spinal meningitis, and the form of posterior basic meningitis of children, first described by Gee and Barlow, all of which are described in other sections, there is a large group of cases of so-called simple meningitis which occur under different circumstances. Such cases may be met with in the course of acute specific diseases, such as variola, measles, scarlet fever, enteric fever, and acute rheumatism; but they are much more commonly associated with pneumonia than with any of these other diseases, and are also met with in connection with septicæmia and pyæmia. Most important causes are also to be found in simple, purulent, and especially obstructive inflammations of the antrum, nasal sinuses, and ethmoid cells; and inflammation of the orbit is responsible for some cases, as is erysipelas of the scalp for others. Diseases of the cranial bones, syphilitic or otherwise, are common causes, in which connection disease of the petrous portion of the temporal bone, secondary to ear disease, plays an important part, though meningitis is less common than is the occurrence of cerebral abscess under these conditions. Injuries to the head often cause this variety of meningitis, and that even when there have been no external evidences of such injuries. Insolation occasionally causes it. Cases are met with in association with Bright's disease, and gout is also said to be a cause. Over-study has been regarded as a direct cause of meningitis, but it is probably only one factor in the etiology of such cases, as will be seen when their pathology is under consideration.

Morbid anatomy.—The precise appearances met with depend on the severity of the process, its mode of origin, and the length of time that it had existed before causing death. In the earliest stage the vessels of the pia are injected, and there is soon added to this a milky-looking opacity of the membranes, especially noticeable along the vessels and sulci of the convexity, and it may be also in the interpeduncular space at the base. This exudation, which gives rise to the opacity, next becomes purulent in patches, which ultimately coalesce, so that when the convexity of the brain is affected the convolutions may be concealed by a yellowish white exudation, or green purulent matter, which is as a rule in the subarachnoid space, and only rarely between the dura and arachnoid. In cases of still longer standing there may be caseation of this purulent exudation. When the base is affected, the interpeduncular space may be filled with this exudation, which may spread to the under surface of the cerebellum. The choroid plexuses sometimes become infiltrated, and the ventricles may contain turbid fluid, or even pus. The pia-arachnoid over the surface of the brain is much thickened, and may be soft and gelatinous, or firm, according to the nature of the exudation. The underlying convolutions may be softened and infiltrated by leucocytes, and it may be impossible to remove the pia mater without tearing the surface of the cortex. Thrombosis of one or other of the cerebral sinuses may be present.

Pathology.—Inflammation of the pia-arachnoid shows little tendency to the formation of lymph, as compared with the frequency with which pus is met with. Symptoms referable to the cranial nerves usually depend on extension of the inflammatory process to them, while other symptoms are in large measure due to the irritation and pressure effects of the exudation on the nerve elements, and the more general increase of intracranial pressure caused by effusion into the ventricles. Another way in which symptoms may be produced, however, is by micro-organisms, whether these be the cause of the affection or not. The cerebro-spinal meninges appear to be a peculiarly favourable place for the multiplication of micro-organisms, which may produce symptoms in meningitis, either by their local irritative effects, or by their toxic products becoming absorbed. It must not, however, be inferred from this that all organisms found in meningitis, or their toxic products, are of necessity the cause of the affection. The organism that has been most commonly found in the condition has been the pneumococcus, even if we only take into consideration the cases in which pneumonia has not existed in conjunction with the meningeal affection. But in addition to this and other organisms that are capable of generating the disease, other varieties may be found in the same case, which, while having no share in the production of the malady, nevertheless play an important part in the course of the morbid process, and yield manifestations by setting up a process of secondary infection. Then, again, many cases of meningitis which appear to be in no way related to micro-organisms, such as those following injury without external wound, over-study, and the like, may in reality prove to be closely associated with them. Thus an injury may induce a state of the meninges suitable for the multiplication of organisms, which in their turn become responsible for the graver manifestations of the disease, while over-study and similar conditions may lower the general resistive power of the subject against their attacks.

There is evidence which makes it probable that the toxines of conditions like measles and scarlet fever can cause meningitis by their direct action; but, on the other hand, attempts to produce the affection in animals, by the inoculation of micro-organisms, have only succeeded where the injections have been made directly into the cerebro-spinal cavity.

Symptoms.—After what has been said with regard to the etiology, it will be readily understood that the symptoms due to a meningitis may be masked or hopelessly confused by those consequent on the disease or injury to which the meningeal affection is secondary, but other cases present a more or less typical clinical picture, by which the condition can be recognised. As a full description of the symptoms met with in tuberculous meningitis will be given later, and as there is a good deal of resemblance between the manifestations of that affection and the other forms of leptomeningitis, a short sketch of the clinical features of the non-tuberculous varieties must suffice here.

The symptoms usually begin quite suddenly without any prodromal stage, but sometimes they are much more insidious and indefinite. Rigors commonly occur at the onset in adults, but are less frequent in children. Headache is almost always present, and is usually the first symptom to attract attention; the seat of the headache varies, and exacerbations of it occur from time to time which may cause the patient to shriek with pain. Vertigo is an early but not a frequent symptom, while vomiting may occur once or more at this stage of the disease. The muscles of the neck may become stiff and the head retracted, both of which are signs of the greatest importance, indicating affection of the meninges of the posterior fossa of the skull, and they are often the earliest unequivocal evidences of the nature of the disease. The stiffness may extend to the muscles of the back and to the limbs. The pupils are as a rule contracted, there is intolerance of light and extreme sensitiveness to sounds. There may be maniacal excitement in the initial stage, or mental disturbance may be absent until the later stages, when the delirium may be maniacal, but depression is more common than exaltation. The temperature is raised, it may reach 104° F. within the first few days, and is very irregular. The pulse is as a rule rapid, and may also be irregular.

Such, briefly, are the phenomena which characterise the early or irritation stage of a meningitis. As the stage of pressure develops there is increasing somnolence, passing on to coma. The pupils are dilated, sluggish, and it may be unequal; ptosis or strabismus may occur, and nystagmus is sometimes present. Paresis of the muscles of the face may result from involvement of the seventh cranial nerve, or there may be anaesthesia through implication of the fifth, and trophic changes in the cornea have resulted when the Gasserian ganglion has been affected. Optic neuritis is not infrequently met with, when the chief mischief is at the base of the brain, but it is rare when the convexity of the organ is alone, or chiefly affected. Other cranial nerves are sometimes, though most rarely involved. Epileptiform convulsions may recur frequently, or there may be more limited spasm, as of a limb, or twitching of individual muscles. So, too, paralysis of the limbs on one side, with or without affection of the face, may occur. Constipation is usually present, and the abdomen is often tense and concave. Towards the end there is loss of control over the sphincters. The temperature remains above 102° F., and the pulse becomes slow and irregular. The duration of this form of meningitis may be only a few days, or even forty-eight hours in severe cases, and rarely exceeds a week or ten days, though some cases are prolonged beyond this time.

Diagnosis.—The diagnosis is comparatively obvious in some cases, while in others it is exceedingly difficult. Acute diseases, such as enteric fever, pyæmia, general tuberculosis, and the like, may be attended with cerebral symptoms, easily mistaken for meningitis; and, on the other hand, in conditions like erysipelas and pneumonia, it is exceedingly difficult to say whether there is more than the symptomatic delirium present. Enteric fever is usually more gradual in onset, and cerebral symptoms do not come on until later in the course of the illness than in meningitis, and the characteristic temperature curve, nature of the stools, enlargement of the spleen, rose-coloured spots, all serve to distinguish it from the cerebral affection. In the case of pyæmia, some cause for the condition will probably be discovered; there are frequent rigors, and there may be subcutaneous hæmorrhages and swelling of joints, in addition to which evidences of metastatic abscesses may be forthcoming. Pyæmia, consequent on septic thrombosis of a lateral sinus, in connection with otorrhœa, may simulate meningitis so closely that the autopsy alone reveals the true nature of the case. Further, there is a class of cases in which, during life, symptoms exist which appear to point clearly to the existence of meningitis, but in which, post-mortem, beyond hyperæmia, or similar slight changes of doubtful significance, nothing is found to account for the clinical phenomena.

In attempting to distinguish the form of meningitis present, every care must be exercised in searching for a possible cause, which, if discovered, will usually aid greatly in deciding this question. The tuberculous variety can be further distinguished in some instances by the detection of tubercles in the choroid on ophthalmoscopic examination, or by the discovery of tubercle bacilli in fluid drawn from the subdural space by Quincke's lumbar puncture. Negative results from cover-glass preparations stained for tubercle bacilli are of no value; inoculation experiments in guinea-pigs can alone be relied on, as such experiments have yielded positive results when a large number of cover-glass preparations had failed to do so.

Prognosis.—The disease is almost certain to terminate in death; but, allowing for the possibilities of mistaken diagnosis, there still remain a few cases in which there has been recovery from simple meningitis secondary to adjacent disease, or traumatic in origin. Recovery from purulent meningitis never occurs; but Gowers speaks of recovery in two instances, where there were distinct symptoms of meningitis in post-puerperal septicæmia.

Treatment.—The patient must be kept absolutely at rest in bed, in a darkened room which is cool and well ventilated. Anything calculated to cause mental excitement or physical unrest must be avoided. The head and shoulders should be raised and cold applied to the head, from which the hair has been shaved, if possible. In young full-blooded persons it may be advisable to practise local depletion, by means of leeches applied behind the ears or on the temples, or even general depletion may be had recourse to with advantage in some cases. With the same object in view, active purgation may be employed, and in any case of meningitis it is essential that the bowels should be kept freely open.

General counter-irritation of the scalp is to be deprecated, but a blister, or the thermo-cautery lightly applied to the occiput, is often useful, and probably does not increase to any extent the amount of suffering of the patient.

The exercise of much discretion is needed with regard to the administration of opiates, as it is imperative that they should be given for the relief of pain in some cases, while in others there is no such urgency, in which case such drugs are best avoided in that their effects mask the symptoms produced by the meningitis. Subcutaneous injections of small doses of morphine, or enemata of chloral, are the best measures to adopt when anodynes are called for. Before either of these drugs are used, antipyrine, phenacetin, and similar remedies should be tried.

Vomiting is best treated by giving the patient ice to suck, and by the application of a mustard plaster to the epigastrium, and another to the back of the neck. Hydrocyanic acid and bismuth by the mouth may also be tried, and when these measures fail, small doses of cocaine may allay the vomiting. Of drugs that have been supposed to influence the inflammatory process, mercury is the most useful. By means of inunction, the patient should be brought under the influence of the drug as quickly as possible, and kept in the condition in which the gums are just "touched." Of other drugs that have been extolled, iodide of potassium and perchloride of iron deserve mention; in the event of the meningitis being of syphilitic origin, the former drug would be of great advantage, while the latter appears to have a distinct influence in septicæmic cases. Quincke's lumbar puncture has been successfully employed in the treatment of some cases, as has trephining and drainage.

Where disease of the ear or antrum or any similar cause can be detected, it must be actively dealt with by a surgeon. Prophylactic measures consist in careful attention to ear disease, inflammatory conditions of the antrum, nasal sinuses, ethmoid cells, and the like, and rigid antiseptic treatment of all injuries of the skull.

J. RISIEN RUSSELL.

TUBERCULOUS MENINGITIS.

This disease is a form of acute tuberculosis in which the membranes of the brain, and it may be of the spinal cord also, are attacked, and in which an excess of cerebro-spinal fluid, with dilatation of the lateral ventricles of the brain, results secondarily.

The first full description of the clinical symptoms of the affection which we possess is by Robert Whytt of Edinburgh, who in 1768 published an account of what he called dropsy of the brain. "Acute hydrocephalus" is the name by which it was generally known at the beginning of the present century, and is even now not infrequently used to denote the affection.

Although recognised clinically so long ago, it was not until 1830 that its pathology became known, when Papavoine showed that the morbid process consisted in the deposition of tubercles in the membranes, and looked on the excess of fluid in the ventricles as of minor importance. Bricheteau, however, first suggested that the condition should be called tubercular meningitis; and in that evidences of inflammation are usually present, in conjunction with the tuberculous deposits, the name has been approved of, and is that now in general use, except that the modern tendency has been to substitute the term "tuberculous" for "tubercular."

Etiology.—While many conditions play their part in rendering the subject liable to this affection, or in evoking its manifestations in one so predisposed, the essential cause is an infection of the cerebral meninges by means of the tubercle bacillus, the process being always secondary to previously existing tuberculous disease in some other organ. In the majority of post-mortem examinations made in this condition, some primary source of tuberculous infection is detectable elsewhere in the body, especially in the lungs and pleuræ, the bronchial and mesenteric glands, bones, joints, genito-urinary apparatus or brain; and even where no such primary focus is discovered, we are scarcely justified in assuming that the process is primary in the cerebral meninges, for a tuberculous lesion in the bones, middle ear, or some other part, may easily escape observation, unless a very exhaustive search is made. Of the factors which render the patient liable to the disease, a hereditary tendency is not the least important. As a rule, cases that are met with have a family history of tuberculosis, either pulmonary or infantile; and many children of the same family are often attacked at about the same age. On the other hand, there may be no such family history, and it must be remembered that the modern tendency is to regard tuberculous disease as due to the introduction of the virus from without, especially in the food. The disease most frequently occurs in children between the ages of 2 and 10 years, but has been met with by Guersant in a child 6 weeks old, and is not infrequently seen in early adult life. It seems especially prone to occur at the period of the first dentition. A few cases occur during the middle period of life, but after that it is extremely rare. Accessory factors, which play a part in the etiology of the disease by lowering the physical condition, and thus rendering the patient less resistant to the tuberculous process, are to be found in impure air, imperfect or insufficient food, and want of exercise. Well-nourished children may, however, fall victims to the disease.

Besides these, there are conditions which appear to act much in the same way, or as direct excitants, and though met with rarely, require consideration. A blow on the head seems to act as a determining factor in some cases, and acute diseases, such as diarrhœa, enteric fever, whooping-cough, broncho-pneumonia, and the like, may all be followed by an attack of this form of meningitis. Foremost amongst such conditions is measles, which appears to have a peculiar tendency to excite tuberculosis. Brain work probably stands in the same relation to the affection as many of the other conditions just mentioned, and, like them, can in no sense be regarded as the cause of the disease, which, as we have seen, depends on a process of tuberculous auto-infection. Surgical interference with tuberculous disease exceptionally leads to dissemination of the virus and consequent meningitis.

Morbid anatomy.—Two distinct conditions are met with on examination of the brain after death, the one being a deposition of miliary tubercles, in the form of minute grey dots, sometimes very difficult to see, or, if caseating, then opaque yellowish in colour, and the other an inflammatory exudation. Sometimes the miliary tubercles are plentiful, and very little lymph is seen, while in other cases the tubercles are scarce and the exudation abundant, gelatinous, and opaque; but there is rarely anything that can be regarded as pus. Tubercle bacilli have been found in the membranes even when no miliary tubercles could be seen with the unaided eye. The most pronounced lesions are usually met with at the base of the brain, though in exceptional cases the convexity suffers most. When the base is chiefly affected, the lymph is found occupying the triangular space bounded by the crura cerebri and optic tracts, and also covering the ventral surface of the pons and medulla, and it may be extending down the spinal meninges. The exudation may spread along the fissure of Sylvius and the neighbouring sulci, until the greater part of the convexity of one or both hemispheres is covered; usually, however, it does not spread far along the fissure of Sylvius, and the membranes over the convexity of the hemispheres may be quite natural in appearance or unduly dry looking. The miliary tubercles are most numerous, as a rule, along the course of the large blood vessels, and consequently occur chiefly in the sulci and furrows on the surface of the brain, along the fissure of Sylvius, at the optic chiasma, and over the surface of the pons, medulla, and cerebellum. They may also spread along the choroid plexuses, which are engorged. The dura may also be the seat of miliary tubercles. Small hæmorrhages occur in the pia and in the brain substance, which may also show inflammatory changes and the presence of tubercle on microscopical examination. The convolutions on the convexity of the hemisphere may be much flattened by pressure consequent on the dilatation of the lateral ventricles, which contain a large excess of cerebro-spinal fluid, which may be turbid or blood-stained. Thrombosis of a cerebral vein is exceptionally met with, and still more rarely a similar condition of one of the sinuses.

Pathology.—The relationship between the symptoms, and the changes found post-mortem, is not wholly clear. In the early stages of the disease the inflammatory process in the membranes seems capable of irritating the nerve elements, and exciting them to over-activity. But considerable paralysis may be met with clinically at this stage, while only insignificant changes can be found in the brain to account for this, a condition of things for which no altogether satisfactory explanation has as yet been offered. In the later stages of the affection the paralytic and other concomitant phenomena are best accounted for by the compression of the brain, which results from the effusion into the ventricles and the damage to the nerve centres by the action of the inflammatory process.

The rigidity of muscles which is met with is due to irritation of the cortex, crus, pons, medulla, or, it may be, even the spinal cord, the amount of retraction of the head depending on the degree to which the posterior fossa of the skull is involved. Affection of the cranial nerves is due either to their irritation or destruction by the inflammatory processes at the base; but the face and tongue may be paralysed as a result of a cortical lesion, and such a lesion may also account for inequality of pupils and strabismus.

Symptoms.—In that the patient is most commonly a child, it is convenient to describe the clinical features of the disease as seen in children, and then to point out how its course differs when an adult is attacked.

The child has usually been "ailing" for some weeks or months before any definite manifestations of meningitis are detected. These prodromata consist in alteration in the child's disposition, restlessness at night, emaciation, pallor, gastro-intestinal irregularities, and so on. Such a child is apathetic and dull, becomes easily tired at games, with its toys and at lessons, begins to get irritable and fretful, or becomes peculiarly affectionate. Although during the day it seems to be tired and wants to lie down, it is extremely restless at night, grinds its teeth, starts and wakes as if frightened by dreams, or if there is the slightest noise, and shows great intolerance to a light in the room. It loses flesh, the skin becoming lax, the contour of the limbs is lost, and the muscles feel flabby and soft, while the ribs and other bony processes stand out prominently. In spite of this the face may not show any evidence by the otherwise general emaciation, but the countenance becomes listless and wanting in variety of expression, the eyes dull, and the face pale. The little patient begins to show a distaste for its food, it becomes constipated, or constipation alternates with attacks of diarrhœa, and there may be slight elevation of temperature. The child may only remain in this condition for about a fortnight, or it may linger on for as much as six months before anything further happens; whereas, directly the more certain invasion of the disease manifests itself, we can predict with comparative certainty that the duration of life will not be more than two to four weeks, and usually three. Before these more definite and rapidly progressive symptoms manifest themselves, however, there may be an intermediate period, calculated to raise false hopes, in which the child begins to improve in health and spirits.

One of two symptoms generally usher in the stage of the disease which so rapidly progresses to a fatal termination, vomiting or convulsions. Of these the former is the more common, and may occur spontaneously or after taking food. It may only continue for a few days, or may last a week, or in exceptional cases it may not occur at all. When convulsions occur they may be epileptiform in character, or more of the nature of tonic rigidity, or the attacks may simply consist in temporary loss of consciousness.

It is now that the peculiar "hydrocephalic cry," so much insisted on by Trousseau, is most commonly heard, though it may not occur until towards the end of the disease. It is a single shriek, as of one in terror, which may be repeated every few minutes or at long intervals. Headache is seldom absent at this stage of the disease, the child evidencing this by crying out and by constantly putting its hands to its head. At first the mental faculties remain clear, but after some days they begin to become blunted; the child becomes dull and apathetic, and delirium gradually comes on and continues, it may be with intermissions, until coma supervenes.

The pupils are usually contracted during this stage, especially when there is intolerance of light, and strabismus is frequently present. The temperature gradually rises to about 103° in the evening, and may be 101° in the morning, or but little above normal at this time. The pulse is first quick as a rule, and becomes slow, and it may be irregular, later; variations in speed occur, slight excitement being sufficient to increase the rate. The respirations are rarely altered. The tongue may be clean or slightly furred, and the bowels are almost always constipated. Such are the symptoms which characterise what is known as the stage of irritation, which continues for about a week, as a rule, and is followed by the next stage, which is known as the paralytic or pressure stage.

The child no longer notices what is going on around it, and consciousness is gradually lost, or somewhat abruptly so in exceptional cases. The knees are drawn up on to the abdomen, which is retracted and boat-shaped, and the head may be slightly drawn back by spasm in the muscles at the posterior part of the neck, though this is rarely marked or continuous. The limbs on one side may be in a similar state of spasm, or there may be convulsions. Ophthalmoscopic examination may reveal optic neuritis, which is slight in most cases, and tubercles are seen occasionally in the choroid. The pupils may be unequal, or they may be equally dilated and insensitive; perhaps one eyelid droops, or there is strabismus. Paresis of one side of the face is less common, but occurs, and an arm or leg may be similarly affected, or there may be paresis of both limbs on one side. So, too, the syndrome of Weber may be met with, consequent on a lesion of the inferior and internal part of the crus involving the third nerve, and consisting in paralysis of that nerve on one side, and the limbs, face, and tongue on the opposite side.

Coma deepens and the evacuations are passed under the patient. The bowels continue constipated, as a rule, but there is no longer vomiting. The tongue either continues moist, or becomes dry. The temperature may be subnormal, or it may rise to 101° F. The pulse is slow, and may be irregular; and respirations also become irregular and present the "Cheyne-Stokes" rhythm, or an abortive form of this type of respiration. The appearance of the patient varies; one or both cheeks may be flushed, or the face may be pale, but any disturbance of the child will as a rule cause the whole face to become red.

A similar condition of things is responsible for the phenomenon known as the *tache cérébrale*, which consists in a red line of unusual breadth and duration, which follows, more quickly than in health, the drawing of the finger nail lightly over the surface of some part of the body.

A convulsion may immediately precede death, or there may be no alteration in the patient's condition, other than that of the passage from coma to death. Bedsores may form and hasten the fatal termination. A curious improvement sometimes takes place a day or two before death, even consciousness being restored, but the pulse rarely shares in the general improvement, and it often becomes very quick during the last few days, or it may remain slow to the end. Hyperpyrexia may result, or, on the other hand, a markedly subnormal temperature may be met with.

In adults the symptoms of tuberculous meningitis are in the main the same as in children. Headache, vomiting, and affection of cranial nerves are much the same; delirium is much more common, and comes on earlier, while general convulsions are uncommon. The attack, though seldom preceded by prodromata, usually begins insidiously, and in young women symptoms of a hysterical character occur at this stage. The onset may, however, be more or less abrupt. In some cases the first symptom is a well-marked paralysis of some part; thus a squint may appear, or a monoplegia or a hemiplegia may come on in the course of a few days, with or without convulsions, and if on the right side may be accompanied by aphasia. Wasting to any marked degree does not occur in the adult, except where it is due to pre-existing disease, such as phthisis, the physical signs of which may be so slight as to escape detection.

The duration of the disease may be much the same as in the child, or it may be longer, while it often appears shorter, possibly owing to the earlier symptoms being masked by the disease to which the meningeal affection is secondary.

Diagnosis.—The condition with which tuberculous meningitis is most commonly confounded is enteric fever. It may be impossible to make a positive diagnosis during the first few days, but, as well-marked cerebral symptoms develop, the recognition of the condition is not difficult. The opposite mistake of taking typhoid fever for tuberculous meningitis is more common, and occurs when the former affection is attended with vomiting, constipation, irregular pupils, strabismus, headache, delirium, and coma. The pulse is more rapid and seldom irregular in enteric, and the temperature curve is more regular. The presence of optic neuritis would weigh largely in favour of tuberculous meningitis, while the detection of tubercle in the choroid would place the diagnosis beyond question. A search for tuberculous disease in other organs may result in its discovery, and thus materially aid in the diagnosis. In all such doubtful cases Widal's serum test for enteric fever should, of course, be employed, the details of which will be found described in the account of Enteric Fever.

In young children, catarrhal febricula may similarly be mistaken for meningitis, owing to headache and subsequent delirium, in reality accounted for by the pyrexia present being regarded as indications that there is cerebral disease. When these conditions result in the course of a general disease, headache is no longer present after delirium begins (Jenner), while in meningitis both symptoms exist at the same time. The presence of optic neuritis, or of limited convulsions or paralysis, point to cerebral disease.

Symptoms like those due to meningitis sometimes result from ear disease, and end fatally without any affection of the meninges being found. There may even be marked optic neuritis and strabismus in some of these cases, so that a correct diagnosis may be quite impossible. Acute double otitis in children is especially liable to present this difficulty.

Hydrocephaloid disease, or spurious hydrocephalus, as it has also been called, was formerly frequently mistaken for tuberculous meningitis. The condition appears to depend on anæmia of the brain, and one of its chief causes is no longer in existence, in that it consisted in the practice of bleeding children whose vitality was already depressed to a great degree. Diarrhœa, insufficient and improper food, and deleterious hygienic surroundings, also play their part in its causation. In this condition, which is confined to young children, while there may be feverishness, irritability, and restlessness at the outset, such symptoms do not continue long enough to give rise to any real difficulty in diagnosis. The face is pale, the fontanelle depressed, somnolence passing on to coma, while all local cerebral symptoms, either spasmodic or paralytic, are absent.

Apart from the fact that tuberculous tumours and meningitis may co-exist, intracranial tumours of rapid growth may be difficult to distinguish from meningitis; especially is this so in tumours so situated as to run a latent course, until they have reached a considerable size. Paralysis in the limbs develops gradually, as a rule, in tumour, while, if present in meningitis, it is usually sudden in onset. Though optic neuritis may be present in both, it is always only slight in meningitis, so that pronounced optic neuritis is strongly in favour of the existence of tumour. Sometimes there is an absence of all reliable information on which to depend for a diagnosis at first, and it becomes necessary to wait on the subsequent course of the disease. The sudden appearance of paralysis early in the case is in favour of meningitis, while a paralysis of slow development at the same stage suggests tumour. If the patient continues to get worse, without rise of temperature and without consciousness being lost, and more than a fortnight has elapsed since the first symptoms were noted, the probabilities are very strongly in favour of tumour.

The more rapid course and the absence of rise of temperature serve to distinguish meningeal hæmorrhage from meningitis, but sometimes, when the symptoms of the occurrence of the hæmorrhage are not developed quite suddenly, there may be considerable difficulty in diagnosis.

Rheumatism of the muscles at the back of the neck, causing retraction of the head, may be attended by elevation of temperature, and thus simulate meningitis, but there is more pain in the muscles and there are no cerebral symptoms. According to Money, similar rigidity in these muscles may result from intestinal disturbances and tender cervical glands.

Cases of tetany are to be distinguished from cases of tuberculous meningitis by the fact that they occur between the ages of 1 and 3 years, while the meningeal affection is more commonly met with between the ages of 3 and 5 years. The long premonitory stage of tuberculous meningitis is wanting in tetany, while if spasm is present in meningitis it does not long resemble that of tetany, and is not repeated with the same constancy or in the same order. The pulse is quick in tetany, and neither presents the slowness nor the irregularity met with in tuberculous meningitis. Headache and vomiting are much less intense. Paralysis of cranial nerves, inequality of pupils, or the presence of optic neuritis, would leave no room for doubt that the meningeal affection was present.

Hysteria is sometimes mistaken for tuberculous meningitis, but more commonly the graver affection is mistaken for that of functional nature in adults and later childhood, especially in females. Sometimes there is no justification for the mistake, but in other cases there are the greatest possible difficulties in diagnosis. In every case where this doubt arises, a careful search must be made for the slightest indication of organic mischief, and we must not allow the presence of hysterical symptoms to prevent us from giving due weight to the significance of evidences of organic mischief, no matter how slight these may be. Rise of temperature, optic neuritis, inequality of pupils, divergent strabismus, nystagmus, and convulsions beginning locally, are most valuable indications of meningitis as opposed to hysteria. The pupils when altered in hysteria are spasmodically contracted, and strabismus if present is convergent, as a rule, though I have once seen conjugate turning of the eyes to one side in this affection.

Quincke's lumbar puncture of the subarachnoid sac, between the third and fourth lumbar vertebræ, may be employed as a means of diagnosis, both as regards the existence of meningitis and as regards its nature. In this connection it must be remembered that in searching for tubercle bacilli, negative results by staining methods cannot be relied on, as inoculation experiments in animals have yielded positive results, where large numbers of cover-glass preparations had been previously examined without tubercle bacilli being found. In searching for bacilli by staining methods, the fluid removed should, if possible, be placed in a centrifugal machine, and cover-glass preparations made from the sediment thus obtained.

Prognosis.—In few diseases is the prognosis so grave as in tuberculous meningitis. Cases of recovery from what has been diagnosed as tuberculous meningitis occur, but are open to the objection that there is no means of verifying a diagnosis which may possibly have been erroneous. Fürbringer has, however, practised Quincke's lumbar puncture in a man who recovered, and whose cerebro-spinal fluid was turbid and contained tubercle bacilli. Evidences of old basal meningitis have in very exceptional cases been found post-mortem, and have been supposed to indicate the previous existence of tuberculous meningitis from which the patient had recovered; but in the absence of miliary tubercle or caseous nodules, such matting together of the membranes at the base may equally well, and with a greater degree of probability, be accounted for by simple meningitis, especially the posterior basic variety

While we regard the prognosis as so bad in this disease, the mere fact that cases occur in which the diagnosis appears warranted, and yet which terminate in recovery, is a warning to us not to assert too positively that a patient suffering from what we believe to be an attack of tuberculous meningitis cannot get better. The course of the disease supplies the best guide in prognosis; early coma within the first week of illness is the worst sign, while if three weeks have passed without consciousness being lost, the chances of ultimate recovery are much improved.

Treatment.—Hopeless as the condition seems, it is our duty to do everything in our power to avert the usual termination. All the measures that have been found successful in other forms of meningitis must be had recourse to in this form of the disease, keeping clearly before us the possibility of mistaken diagnosis and consequent chances of recovery. The room should be darkened and kept as quiet as possible; the patient should be encouraged to sleep, and should not be disturbed more than is absolutely necessary. The head and shoulders ought to be raised, care being taken not to embarrass the cerebral circulation or respiration by flexion of the neck. Mental excitement and depression must alike be carefully guarded against. Ice should be applied to the head, from which the hair has been removed, and warmth and mustard applied to the extremities, with a view to diminish the amount of blood to the head. Little effect on the morbid process can be hoped for from counter-irritation, which is best avoided, and any general counter-irritation, such as blistering the whole scalp, is to be strongly deprecated.

Easily digestible, nutritious food must be administered every three hours, and when the patient cannot be induced to swallow, nasal feeding or nutrient enemata or suppositories must be substituted. Stimulants are often called for in the last stages of the disease, before which it is better not to give them, as a rule; but the state of the pulse is the best guide as to whether they are wanted or not.

Of drugs, mercury has deservedly gained a good reputation in the treatment of meningitis, and should always be given a prolonged trial even in this form of the disease. The oleate or ointment should be rubbed into the scalp, back of the neck, armpits, or groins every four hours, so as to get the gums "touched" as quickly as possible, after which the inunctions should be repeated at such intervals as prove necessary to keep the patient slightly but distinctly under the influence of the mercury. It is doubtful how much good iodide of potassium is capable of doing; it is certainly not as efficacious as mercury, but may be tried in 5 gr. doses three times a day in children, and three times this quantity in adults.

Tuberculin treatment of these cases has not yielded any more encouraging results than has followed the same treatment of other tuberculous affections, but the temporary amelioration which followed the injections in some cases justifies the hope that better results may possibly attend the use of Koch's new tuberculin.

Quincke's lumbar puncture of the subarachnoid sac has been successful, but can only be of service in those cases in which the foramen of Magendie is not closed. Trephining and drainage has also been followed by recovery in what has been regarded as tuberculous meningitis.

Headache may be relieved by the application of cold to the head or by the administration of antipyrine; and if the patient is very restless, it may be necessary to give sedatives, but of these opium must be studiously avoided. Vomiting is best dealt with by mustard plasters to the epigastrium and back of the neck, and ice or cocaine by the mouth. The bowels should be kept freely open, care being taken not to reduce the patient's strength too much, however.

It may be necessary to reduce the temperature in some instances, in which case cold sponging, packing, and baths are much to be preferred to antipyretic drugs; but recourse has to be had to one or other of these, under some circumstances.

J. RISIEN RUSSELL.

POSTERIOR BASIC MENINGITIS.

This form of meningitis is non-tuberculous, and is chiefly limited to the posterior and central region of the base of the brain. The disease has been chiefly studied by Gee, Barlow, Lees, and Walter Carr.

Etiology.—It is met with most frequently in children less than a year old, and usually between the third and twelfth months. Traumatism and ear disease have been too infrequently met with in these cases to make it probable that either of these is responsible for the condition. Similarly, there is no evidence that the affection is syphilitic in origin. It may be in some way related to epidemic cerebro-spinal meningitis, as has been suggested by Carr, and whether this be so or not, the disease is probably caused by a diplococcus which has been repeatedly found, by Still, in the exudation at the base of the brain in these cases, and which is pathogenetic to guinea-pigs, rabbits, and mice.

Morbid anatomy.—The morbid changes are curiously limited in area at the base of the brain, extending from the optic commissure to the lower end of the medulla oblongata, over which region the pia-arachnoid is much thickened, and the parts are matted together. This thickening is somewhat sharply limited by a well-marked margin on the under surface of the cerebellum, about a half to three quarters of an inch on each side of the middle line. In some cases there is a layer of puriform lymph of variable thickness over the affected area, while in others it is absent. Isolated patches of lymph may also be met with in other situations. The pia mater between the cerebellum and medulla is usually thickened, and thus in many cases the cerebral ventricles are shut off from the spinal subarachnoid space. The third and lateral ventricles are always distended.

Pathology.—The relationship between the predominant early symptoms and the morbid changes at the base of the brain, and those of later development in association with increased intracranial pressure, are sufficiently obvious, in view of what has already been said in this connection when other varieties of meningitis were under consideration. The mode of origin of the hydrocephalus in these cases is a matter of considerable doubt, and three possible theories have been advanced to account for it. The first of these supposes an inflammatory blocking of the foramen of Magendie, the second an inflammatory effusion into the ventricles, and the third a simple dropsical effusion, consequent on obstructed blood flow through the veins of Galen. There are objections to each of these theories, and it seems possible that the same cause is not in operation in all cases. Carr's observations lead him to advocate the view which supposes the condition to depend on venous obstruction, but he thinks it possible that in some of the cases an inflammatory process may be contributory.

Symptoms.—The onset may be sudden or gradual, retraction of the head and vomiting being the most prominent early symptoms. The former of these conditions persists throughout the course of the disease, is much more marked than in tuberculous meningitis, and is sometimes accompanied by general opisthotonos. Vomiting may also occur frequently throughout the course of the illness. Slight twitchings are common, but general convulsions are rare, except at the onset. Stupor and coma supervene comparatively early, but life is preserved for many days, or it may be weeks after this event. A fixed stare may result from retraction of the eyelids, or there may be clonic spasms of the lids. Strabismus is common, but nystagmus and optic neuritis are only rarely met with; the former, however, more commonly than in tuberculous meningitis. In spite of the absence of detectable optic neuritis, the patients are often blind for a time. There is rapid emaciation towards the last, death frequently resulting from inanition. Breathing is often irregular, or Cheyne-Stokes in character. The pulse is rapid but regular, and is never slow in infants, though it may be so in older children. The temperature is normal, with the exception of occasional elevations, but hyperpyrexia frequently precedes death, which usually results in from three to fifteen weeks.

Diagnosis and prognosis.—It is easy to mistake cases of this affection for cases of tuberculous meningitis. The chief points which serve to distinguish the posterior basic variety are the following:—From the time that definite symptoms become evident, to the termination of the case, is shorter in the tuberculous form; and the length of time that supervenes after the patient becomes comatose and the pulse rapid, before death results, is much shorter. The pulse is never slow in the posterior basic form in infants, as in the intermediate stage of the tuberculous variety, and the temperature is often quite normal for long periods. Retraction of the head is rarely so pronounced and never so persistent in tuberculous meningitis, while the extreme retraction of the abdomen so characteristic in this disease is not seen in the simple posterior basic variety. Vomiting often ceases in the later stages, while in the posterior basic form this symptom frequently continues to the end of the illness. Constipation is more common in the tuberculous form. Lumbar puncture is of no value in diagnosis, owing to closure of the foramen of Magendie. Although this is a very grave affection, a fair proportion of cases recover completely. There may be remaining hydrocephalus in some, others are blind—at any rate for a time—some permanently deaf, and others mentally defective. Possibly some of the recoveries are numbered among those of supposed recovery from tuberculous meningitis.

Treatment.—An ice-bag should be applied to the head, and mercurial ointment rubbed into the neck. Iodide of potassium and grey powder or calomel have been administered internally, but with no special advantage. If hydrocephalus is developed, the lateral ventricles should be punctured, and very small quantities of fluid drawn off. Permanent drainage from the ventricle into the subdural space has also been tried, once with success. Operative interference, which has as its object drainage of the fourth ventricle, must be considered in the acute stage as soon as marked pressure symptoms develop, or later if the hydrocephalus appears to be increasing in spite of puncture of the lateral ventricles.

J. RISIEN RUSSELL.

CHRONIC HYDROCEPHALUS.

In this condition, which is also known as dropsy of the brain, there is a large collection of fluid, either between the dura mater and brain, or in the ventricles, so that an external and internal variety of the affection is recognised.

Etiology.—**External chronic hydrocephalus.**—This is, as a rule, a compensatory condition, and depends on the accummulation of fluid between the outer surface of the brain and the inner surface of the skull, to fill up the space that would otherwise be empty in atrophy of the brain, such as results in senile conditions and in cases of arrested development of the organ. Sometimes the collection of fluid is limited to certain regions by adhesions of the pia-arachnoid to the dura; it is then said to be sacculated, a condition which probably depends on previous meningitis. There is still another class of cases, congenital in origin, in which, without obvious diminution in the size of the brain, there is an excess of fluid in the subdural space.

Internal chronic hydrocephalus.—This is a condition which is either congenital or acquired. In the former cases labour may be obstructed by the enlarged head, but more commonly there is nothing to attract attention at birth, or only moderate enlargement of the head then exists, while it subsequently increases in size rapidly. There have been many speculations as to the probable cause of the affection, but nothing definite is known in this connection, except that several members of the same family have been attacked.

The acquired cases are due either to some mechanical hindrance to the return of blood from the intraventricular vessels, by pressure on the veins of Galen by a tumour at the base of the brain or in the third ventricle, or to closure of the foramen of Magendie so that there is no longer any communication between the fluid in the ventricles and that in the cerebro-spinal meninges. Hæmorrhage from an aneurysm of the internal carotid has been responsible for the condition, by closing the foramen by adhesion of the tela choroidea to the floor of the fourth ventricle. I have examined a case in which the hydrocephalus was caused by a tumour obstructing the aqueduct of Sylvius.

In a third group of acquired hydrocephalus the cases appear to arise spontaneously, in that no cause has been discovered to account for them.

Morbid anatomy.—The morbid appearances met with in external hydrocephalus, and in those varieties of acquired internal hydrocephalus secondary to tumour, meningitis, and the like, will be found described in those sections of this work which deal with the primary condition on which the hydrocephalus depends.

In the congenital or primary forms of internal hydrocephalus, the conditions met with post-mortem vary considerably, according to the degree to which the process had advanced before terminating in death. The cranial bones may be as thin and translucent as paper; the fontanelles and sutures widely gaping, with Wormian bones developing in them. The brain may be so flattened that the sulci are obliterated, or shallow grooves alone remain to represent them, and the cerebral substance left, forming as it were the walls of a sac, containing the enormous accumulation of fluid, may not be more than a few millimetres in thickness. All distinction between grey and white matter is lost. The basal ganglia, crura cerebri, pons, optic chiasma and tracts, and other structures at the base of the brain, are much flattened out and increased in breadth in consequence. The lateral ventricles are enormously distended, with a clear limpid fluid, which contains a trace of albumin and certain salts. The ependyma is usually of an opaque white or grey colour, thickened and tougher than normal; the surface may be covered with granulations, and numerous amyloid bodies are often present. The choroid plexuses are engorged, sometimes thickened, but at other times they seem normal. The third ventricle commonly shares the dilatation of the lateral ventricles, and with the foramen of Magendie closed, the fourth ventricle may be transformed into a closed cystic cavity. The aqueduct between the third and fourth ventricles and the foramen of Monro are similarly widely dilated. The white substance of the cerebellum has been found nearly entirely destroyed, the organ being hydrocephalic; and in a case of this kind, recorded by Chiari, the cerebellum occupied a spina bifida cervicalis. But apart from complete displacement of the organ in this way, it is common to find the tonsils and inferior part of the middle lobe of the cerebellum elongated like a cone, which is pushed with the medulla oblongata into the spinal canal. The tissues forming the cone are usually sclerotic; and, even with no such displacement of part of the organ into the spinal canal, the cerebellum has been found in a state of hyperplasia.

Symptoms.—In the congenital form, enlargement of the head is usually the first and most conspicuous symptom, though epileptiform convulsions, or some other evidence of intracranial disturbance, may first attract attention. As the head enlarges, the cranial bones become very thin, and separate more and more from each other, except at the base, like the petals of an opening flower (Trousseau), and the fontanelles and sutures thus become much widened, Wormian bones develop in them, and complete the ossification in arrested cases. The hair is scanty, the surface veins of the scalp stand out prominently, and fluctuation can be elicited. In contrast to the enormous head, the face looks unusually small, and has a sharp chin and angular features. There is exophthalmos with turning down of the eyeballs, so that a considerable amount of sclerotic is exposed above, while a large part of the iris and pupil is hidden by the lower lid; indeed, the condition may be so extreme as to allow nearly the whole of the cornea to be covered by the lower lid, a state of things resulting from depression of the orbital plates by the increased intracranial pressure.

The heavy head rolls about as if the neck muscles had lost all power, and there may be inability to raise the head from the pillow. Mental defect is frequently considerable; but even when the affection is extreme, there may be no such mental failure. All degrees of impairment of motor power are met with in the limbs, from slight loss of power to complete paralysis; the muscles may be atrophic, and the paralysis may be attended with loss of control over the sphincters. In addition to the epileptiform convulsions, which occur in some cases, various spasmodic conditions, either tonic or clonic, are met with in the limbs. There is often nystagmus, and there may be strabismus, while the globes are often rolled from side to side. Optic atrophy and blindness results in extreme cases.

Exceptionally the congenital form becomes arrested, in which case the patient may live to an advanced age, but some mental defect may remain; there may be some spastic paralysis, and epileptic fits may occur. Or such cases have been known to show natural physical and psychical development during many years of their lives, and subsequently develop grave symptoms, simulating those due to other organic diseases of the brain. But the more usual event in congenital hydrocephalus is death, within the first few months of life, from marasmus, or it may be from convulsions or coma.

The symptoms of acquired hydrocephalus resemble those met with in the congenital form. In children whose cranial sutures are not closed, the head enlarges but does not attain the enormous size it so often does in the congenital cases. After the sutures have been closed, they may separate again, and, even in the adult, the same result may come about, owing to thinning of the cranial bones. But, as a rule, the adult form differs from that met with in children, in that the enlargement of the head is wanting. The symptoms are very variable, but one that is rarely absent is headache, and it may be associated with optic neuritis, attacks of somnolence or coma, without focal symptoms; but, on the other hand, the symptoms may be indistinguishable from those occasioned by tumour, including partial hemiplegia, with or without aphasia, and convulsions, which may be unilateral, or there may be pronounced ataxy in some cases. There is also peculiar liability to sudden death, which is an event not uncommonly met with in connection with cerebral tumours. It must be remembered that there is a class of cases in which tumours and acquired hydrocephalus are combined, the latter depending on the former condition.

Diagnosis.—A mere glance at the head in a pronounced case of congenital hydrocephalus is sufficient to decide the nature of the affection; but slighter cases may require a little more care in diagnosis, the condition from which they have to be distinguished being rachitic enlargement of the skull. In hydrocephalus, the skull is globular and the fontanelle tense; while in rickets the head is rather square, and the fontanelle, though large, is not tense. Other evidences of rickets can be found, and there is an absence of mental defect and motor paralysis.

Gowers speaks of another condition which may be confounded with hydrocephalus—slow progressive enlargement of the head, the result of thickening of cranial bones.

In the absence of enlargement of the head, though the disease may be suspected, it cannot be diagnosed with certainty, in that the symptoms to which it gives rise are more commonly produced by other intracranial conditions.

Prognosis.—The prognosis is usually bad; congenital cases as a rule die within the first four or five years, but there is always considerable uncertainty as to what may happen. If the quantity of fluid is moderate, and it does not tend to accumulate rapidly, there is more chance of arrest of the disease, and cases in which such patients reach adult life are not very uncommon. In acquired cases the prognosis depends on the cause, recovery being possible when meningitis is responsible, while there is no such hope in the case of tumour, unless removal of the growth be possible. The apparently idiopathic cases in adults are also singularly fatal.

Treatment.—Little good results from treatment in hydrocephalus as a rule. Drugs are quite incapable of causing absorption of the fluid. Diuretics do but little towards getting rid of some of it, and active purgation, while capable of doing more in this direction, is, as a rule, contra-indicated by the feeble condition of the patient.

In external hydrocephalus, puncture of the membranes has been employed; and in the internal variety the lateral ventricles have been tapped under antiseptic precautions, through a trephine opening from time to time, or continuous drainage has been adopted; but in the majority of cases the results have been far from encouraging. Quincke's procedure of puncturing the subarachnoid sac below the termination of the spinal cord, between the third and fourth lumbar vertebræ, is preferable, in that the fluid can be withdrawn more gradually; thus the danger of collapse is much less. It can, however, only relieve internal hydrocephalus when there is no obstruction to the escape of fluid from the ventricles; therefore, when intracranial pressure is not relieved by this procedure, the advisability of direct puncture of the ventricles has to be considered. Sutherland and Watson Cheyne recommend permanent drainage from the ventricles into the subdural space.

Compression of the head by plaster, the strips crossing at the vertex, and kept in position by another encircling the head, or by means of a broad elastic band, with or without the removal of fluid, may be tried; but the effects must be carefully watched, and any evidence that the brain is being compressed must be the signal to loosen the plaster or bandage.

Hygienic measures, suitable feeding, and tonic or other medicinal treatment, must of course be adopted to suit the exigencies of any given case.

J. RISIEN RUSSELL.

THROMBOSIS OF THE SINUSES AND VEINS.

The writings of von Dusch and Gerhardt did much to direct attention to this condition originally, but important contributions to the subject by many authors have since appeared in this and other countries.

Etiology.—Sinus thrombosis may be simple or infective. The simple or non-infective form is met with in ill-nourished children, especially during the first six months of their existence, and usually supervenes after severe exhausting diarrhœa of some weeks' duration. It also occurs after any exhausting disease, such as long-continued suppuration or specific fevers, both in children and adults; and in the latter it is especially liable to be met with in the final stages of phthisis or cancer. Parturition, particularly where there has been much loss of blood, is responsible for the occurrence of some cases, and the subjects of anæmia and chlorosis, notably young girls, are also liable to this complication. Compression of the sinus by a tumour is capable of bringing about the condition.

The infective or secondary form of thrombosis is more than twice as common as the primary variety, and results from the extension of inflammation to the sinus wall from some neighbouring part. It is met with in connection with disease of the internal ear, caries, injury or fracture of the bones of the skull, and meningitis, especially the tuberculous variety. It is also met with in association with suppuration outside the skull, as in erysipelas, carbuncles, suppurating eczema, and ulcerative processes about the scalp, face, and neck. This form of thrombosis has also been met with in pneumonia, and the pneumococcus has been found in large numbers in the thrombosed sinuses. Other organisms that have been found under similar circumstances are streptococci and bacterium coli.

Morbid anatomy.—The conditions met with vary according to whether the affection is simple or infective. In the former the wall of the sinus is very rarely inflamed, the clot is adherent to it, and may completely or only partly fill the cavity. Recent thrombi are dark red and soft, but as they get older they become pale and friable. In cases secondary to a septic inflammatory process the wall of the sinus is usually inflamed, and the contained clot is broken down and puriform in appearance; added to which, meningitis is commonly present.

Thrombi may have spread into the veins communicating with the sinus; but in any case the veins are engorged, and congestion and œdema of the brain results; so, too, areas of cerebral softening occur, and capillaries rupture and give rise to hæmorrhages. If recovery takes place, atrophy and induration of the affected parts of the cortex remain permanently.

Symptoms.—Clinically the affection is very difficult to recognise, so variable are the symptoms, which may in addition be masked by those due to the condition responsible for the thrombosis.

Primary thrombosis of the longitudinal sinus may be met with post-mortem, without having given rise to any symptoms during life, or there may have been headache, vomiting, convulsions, somnolence, mental apathy and depression, terminating in coma. In other cases there may be local or unilateral convulsions or paralysis, indicating extension of the thrombosis to the veins on one side or its commencement in them. The muscles of the neck and back sometimes become rigid, as do those of the limbs in some cases, while strabismus and tremor of the tongue have been exceptionally noted. In addition to these symptoms, there may be distension of the surface veins, with œdema of the forehead and temples; epistaxis may occur, and in debilitated children the previously depressed fontanelle may become prominent and tense.

When the cavernous sinus is blocked there is headache, a varying degree of proptosis of the eyeball on the side of the lesion, and possibly paralysis of the ocular nerves which run in the wall of the sinus, together with the first division of the fifth. Distension of the veins about the orbit, with œdema of the eyelids and temples, commonly occurs, the retinal veins may be distended, there may be large hæmorrhages, and the optic papilla may be slightly swollen.

The symptoms of lateral sinus thrombosis are similar to those caused by a limited meningitis in this situation. Ear disease is present in most of these cases; but this sinus may become thrombosed, in conjunction with others, from a variety of causes. Dilatation of veins and œdema over the mastoid region may be present.

The symptoms produced by thrombosis of secondary origin are, as a rule, those of septicæmia. The lateral sinus is most commonly affected in this way, the disease spreading from the posterior wall of the middle ear more commonly than from the mastoid cells. There is a sudden appearance of acute symptoms in a person the subject of chronic ear disease, consisting in rigors, rise of temperature, and pain in the neck and occipital region: the temperature may remit or intermit, there may be marked optic neuritis, and delirium or somnolence ushers in coma. Motor symptoms, either in the form of convulsions, rigidity, or paralysis, if present, depend on concomitant meningitis rather than on blocking of the sinus. In lateral sinus thrombosis there may be extension of the clot to the jugular vein, which feels like a cord along the anterior border of the sterno-mastoid muscle. The average duration of such cases is about three weeks, death resulting from pulmonary pyæmia as a rule; indeed, the local symptoms may be so slight that the true condition is not suspected until the lung abscesses develop.

Diagnosis.—In young children the condition known as hydrocephaloid gives rise to precisely the same symptoms as does sinus thrombosis; but if such symptoms persist, in spite of cessation of diarrhœa and improvement in the patient's general condition, the probabilities are in favour of thrombosis. Any focal symptoms make this diagnosis still more probable, while enlargement of surface veins and external œdema make it certain. External œdema and distension of veins are also the only reliable signs of marantic thrombosis in adults, but the condition may be suspected if cerebral symptoms occur in a person who presents no evidences of arterial degeneration or cardiac disease, but in whom one of the known etiological factors of sinus thrombosis is present. In old people, cerebral symptoms of the same kind are more likely to be due to arterial thrombosis.

The chief difficulty in the secondary form is to distinguish the thrombosis from meningitis; reliance has mainly to be placed on the septicæmic symptoms and any external œdema which accompany the former condition. The detection of pyæmic abscesses in the lungs would further point to septic thrombosis as the cause of the cerebral symptoms; while extension of clot into the jugular vein is of similar significance.

Prognosis.—This depends on the nature of the thrombosis, but it is always most grave. Children with marantic thrombosis sometimes recover, as may adults the subjects of anæmia or chlorosis, while in cases of phthisis and cancer no such result can be hoped for. It must always be remembered that though recovery may take place, mental impairment may remain permanently. Secondary thrombosis with septicæmia is always fatal, unless prompt surgical measures are adopted, in which case recovery becomes possible.

Treatment.—Prophylactic treatment is of most importance in primary thrombosis, in combating the conditions calculated to lead to this serious result. Once established, however, the patient must be placed in the position most likely to facilitate the flow of blood in the particular sinus involved. In the absence of diarrhœa, a copious hot-water enema may be absorbed, or intravenous saline injections may be of service. The circulation should be encouraged as much as possible by stimulants, and the patient's strength improved by a tonic line of treatment.

Nothing short of prompt and radical surgical interference is likely to be of any avail in secondary thrombosis. Measures must be taken to eradicate the disease to which the thrombosis is secondary, and the sinus cleared out where this is possible. The lateral sinus has been successfully opened and evacuated in this way, after ligature of the jugular vein. While alone they can be expected to do little to avert a fatal termination in such cases, drugs like quinine, salicylate of soda, or perchloride of iron, are important and useful adjuncts to the surgical treatment.

J. RISIEN RUSSELL.

ENCEPHALITIS

Encephalitis, or cerebritis, is, as its name implies, an inflammation of the brain substance, a condition which may be acute or chronic, while the former variety may or may not be attended by the formation of pus. In the present section we propose to limit ourselves to a consideration of the non-suppurative forms of encephalitis, reserving for subsequent and separate consideration abscess of the brain.

ACUTE ENCEPHALITIS.

There are several varieties of this affection. That which has been longest recognised consists in a diffuse inflammation of a more or less extensive area of the brain, which, as a rule, depends on some local cause. Another variety is that in which the hæmorrhagic element is pronounced, and which depends on some form of infection; this is known as acute hæmorrhagic encephalitis; while when the morbid process affects the grey matter of the third ventricle, and that round the aqueduct of Sylvius, it constitutes Wernicke's polio-encephalitis superior. When in addition to this there is involvement of the grey matter of the floor of the fourth ventricle, it is known as polio-encephalitis inferior; and when the inflammation affects the grey matter of the spinal cord as well as the cerebrum, pons or medulla, the condition is termed polio-encephalo-myelitis.

Etiology.—Encephalitis frequently results from some form of injury to the head, such as a fracture, punctured wound, or concussion. The white matter is more commonly affected than the grey, and the inflamed area may be at the seat of injury, or, owing to contre-coup, the opposite side of the brain may be affected. When the brain has been anchored by adhesion of the meninges, in the process of healing after the operation for the evacuation of a cerebral abscess, sudden movement causes a dragging on the brain, which, according to Macewen, has a similar effect to a blow, and when frequently repeated may produce encephalitis. Caries and osteitis frequently cause inflammation of the brain alone, or more commonly associated with meningitis, and suppuration usually results.

In operations on the brain, in which from some cause attempts to obtain asepsis have failed, an acute, rapidly spreading inflammation occurs; or the process may be more chronic, as seen in cases of hernia cerebri. Caustic agents employed for the treatment of chronic otorrhœa have been known to pass through a perforation of the tegmen and produce a fatal encephalitis.

The brain substance immediately surrounding an area of softening from vascular occlusion, septic in origin, may become markedly inflamed. Acute hæmorrhagic encephalitis is brought about by some infective agent, which has been most commonly derived from the organism of influenza, but which is also frequently supplied by the pneumococcus. Among other infectious processes which are responsible for its occurrence, are epidemic cerebro-spinal meningitis, enteric fever, scarlet fever, typhus, measles, and small-pox. So, too, it has been met with in the puerperal state, and in connection with ulcerative endocarditis. Exceptional causes that have been assigned are erythema nodosum, anæmia, and excessive heat, while in some cases no cause can be discovered. Children and those under 20 are most commonly affected, and more women suffer than men.

The other varieties of this affection depend on essentially the same causative factors, but alcohol plays an important part in the etiology of the form known as superior polio-encephalitis. Indeed, alcoholism is the most common cause, and the affection may in some cases be associated with the polyneuritis which results from this poison; but the same infective agents which are responsible for acute hæmorrhagic encephalitis also cause this affection, notably the organisms of influenza, pneumonia, and diphtheria—and cases of intoxication have been met with in which lead, sulphurous acid, and carbonic oxide have appeared to be the cause.

Morbid anatomy.—Red softening is the condition which results from acute inflammation of the brain, the red colour depending on hæmorrhagic extravasations, and on engorgement of vessels. The affected area is swollen, œdematous, and soft, and the microscope reveals, in addition to engorged vessels and minute hæmorrhages, collections of leucocytes around the vessels. The neurons are seen in various stages of degeneration, the axons have fusiform enlargements in their course, and afterwards become disintegrated, while the cells first pass through a stage of cloudy swelling and then degenerate, or atrophy, and become pigmented. At a later stage, on macroscopic examination, cavities are seen which contain a yellowish fatty-looking emulsion, the colour of which depends on the remains of blood pigment; while still later a clear fluid may alone remain in the cavity. Ultimately, a cicatrix results with or without a little collection of hæmatoidin crystals and débris in its centre.

The condition resulting from acute specific processes, while sometimes presenting foci large enough to be detected by the unaided eye, as a rule can only be detected microscopically, and micro-organisms may be seen separately or in colonies around the vessels, or apart from them.

In the various forms of polio-encephalitis the inflammatory condition is characterised by congestion, engorgement of vessels, capillary emboli, infiltration of the tissues with leucocytes, dilatation of lymph spaces, and minute and even large hæmorrhages. A varying degree of destruction of the nerve elements is secondary to these vascular changes, and there is proliferation of the glia elements when the disease has been of sufficiently long duration to allow of this. In cases following influenza and similar diseases, the characteristic organism has been found in the affected areas of the central nervous system.

Pathology.—Although it is abundantly evident that the forms of polio-encephalitis are caused by infective agents, it is not yet known whether the toxines are alone effective, or whether the presence of the organism is essential to the production of the lesions. In some cases, however, no lesion can be detected, which makes it probable that toxines are capable of producing disturbances, resulting in over-activity and diminished activity of nerve centres depending on chemical changes, without demonstrable lesions of the nerve elements, by such methods of examination as have hitherto been employed in their investigation. Disordered vasomotor action may play an important part in such cases; the probability of this is strikingly suggested by the fact that when lesions are met with they are primarily vascular.

Another question that has yet to be decided is, whether the changes met with may be the manifestations of a primary infection, as Strümpell and others have contended. In epidemics of polio-myelitis, many cases of polio-encephalitis have been met with, which suggests the possibility that the same infective agent may be responsible for both conditions; moreover, polio-myelitis and polio-encephalitis have occurred simultaneously in the same individual.

The manifestations of the disease, as a rule, depend on the morbid conditions met with; but in cases in which lesions can only be detected in the mid and hind brain, and yet there have been psychical and other indications of cerebral disturbance, it is probable that these depend on cortical changes too slight to be discerned by the methods hitherto employed in their investigation.

Symptoms.—We possess no very satisfactory knowledge of what symptoms can be regarded as characteristic of acute non-suppurative encephalitis, in that it is so rare to meet with it uncomplicated by any other condition. Headache occurs most commonly, vomiting not nearly so frequently, and optic neuritis rarely. There may be delirium and general convulsions, or, if the sensori-motor cortex happens to be involved, there may be convulsions, or paralysis of one side of the body, or even of more limited distribution. When of traumatic origin, the symptoms of this affection do not come on for two or three days after the injury, and can thus be distinguished from the immediate effects of a wound of the brain.

Acute hæmorrhagic encephalitis.—The recognition of this affection as a distinct entity is primarily due to the investigation of Strümpell, but the observations of Fürbringer, Leichtenstern, and Oppenheim have done much to establish it on the firm basis on which it now stands. A somewhat anomalous case that I have examined leads me to suspect that some of the cases that are regarded as of this nature in reality depend on multiple thrombosis of minute vessels.

The onset is, as a rule, sudden, headache being a prominent and severe symptom. Vomiting is less common, and may, or may not, be associated with vertigo. The temperature rises, and may reach 104° or 105° F., rigors sometimes preceding this rise, or there may be no elevation of temperature throughout the course of the malady.

The onset may be so sudden that the patient at once becomes semi-conscious, and this state may quickly pass on to coma; but whether this be so or not, the mental processes soon become dull, and marked mental obfuscation is followed by stupor. On the other hand, the onset may be slower, headache being accompanied by irritability, thirst, and loss of appetite; and though of rare occurrence, there may be delirium, or an epileptiform attack may be one of the earliest indications of the disease. Apart from this, it is rare to meet with any evidence of over-action, such as spasms or convulsions.

Paralytic symptoms are the rule, though some cases run their course without any such manifestations. When present they consist in monoplegias, or hemiplegias of slow onset and of different degrees of intensity, or paralysis of much wider range, even including all four extremities, the arm, however, nearly always suffering more than the leg. No sensory impairment has been detected, when the patient's mental condition has allowed of this being tested. There may be articulatory speech defect, or motor or sensory aphasia; motor aphasia, or some form of paraphasia, is most commonly met with, and the disease may be ushered in by speechlessness. The pupils are, as a rule, equal, and respond to light. Optic neuritis is met with in a considerable proportion of cases.

The tendon-jerks are diminished, while the superficial reflexes are preserved, and the sphincters are intact until the later stages of the affection, when there may be loss of control over them. Inco-ordination, evidence of implication of the nuclei of various cranial nerves, or bulbar symptoms, may each result according as the cerebellum, pons, or medulla becomes involved by the morbid process. Respirations are rapid and shallow, and may become Cheyne-Stokes in rhythm as the disease progresses. The pulse is quick and feeble. The disease may progress steadily until death closes the scene, but more commonly there are periods of remission, in which consciousness may return, and the patient may become restless and wakeful for a time, and then relapse into the former condition, or one of deeper coma.

Superior polio-encephalitis.—This condition was first described by Wernicke, who, with other of the earlier writers on the subject, regarded it as the result of alcoholism, and as invariably terminating fatally, both of which views have since been proved to be erroneous, though Jacobäus still contends that the affection is a complication of polyneuritis of alcoholic origin.

Like acute hæmorrhagic encephalitis, this condition is, as a rule, sudden in onset, but occasionally there are premonitory symptoms. In alcoholic cases, more especially, symptoms similar to those met with in delirium tremens may occur, while in other cases it is more common to meet with headache, vomiting, vertigo, diplopia, stiffness of the neck, unsteady gait, or some such symptoms in the earliest stage.

Paralytic symptoms occur early, and consist in ptosis and paralysis of various of the external ocular muscles. Complete ophthalmoplegia externa may result, but the internal muscles usually escape, or are only very slightly affected. The facial nucleus is very much less commonly affected; but bilateral and unilateral facial paralysis have both been met with. Optic neuritis with hæmorrhages generally occurs.

Motor weakness of the extremities may be only slight, or more pronounced, and the patient's gait shows a varying degree of inco-ordination, the unsteadiness and reeling being sometimes most pronounced. The tendon-jerks are abolished, or much diminished. Sensation is unaffected.

The mental faculties become blunted, and coma supervenes and continues until death, which usually results in the second week; or loss of consciousness may be less pronounced, and the patient may recover, or the condition may become chronic.

Apart from occasional slight rises of temperature in the early stages, these cases run an afebrile course. The pulse is rapid and becomes progressively more and more feeble.

Inferior polio-encephalitis.—This condition is essentially the same as that just described, the only difference being the seat of the lesion, the superior variety affecting the ocular nuclei chiefly, while in this the bulbar nuclei are also involved. When the disease is limited to the medulla, it is called polio-myelitis bulbi; while, when it not only involves the ocular and bulbar nuclei, but also the anterior horns of the spinal cord, it is known as polio-encephalo-myelitis.

Except in the cases in which the disease is limited to the medulla oblongata, progressive paralysis of the external ocular muscles is the first symptom, and in the variety of the affection which is most common, namely, polio-encephalo-myelitis, evidence of spread of the disease to other cranial nerve nuclei, including those of the medulla, may next be seen in the presence of paresis of the facial muscles, the tongue, and palate, with loss of reflex irritability of the latter, difficulty in swallowing, rise of temperature, tachycardia, and dyspnœa. Or these symptoms may be preceded by those of spinal origin, consisting in paralysis with atrophy of groups of muscles in the extremities, as is seen in acute anterior polio-myelitis.

In polio-myelitis bulbi the onset may be sudden or preceded by vomiting, giddiness, and subjective sensations about the heart. Evidence of paralysis of the pharynx, palate, and tongue are early symptoms, and fainting attacks, tachycardia, and difficulty of breathing, which is so great at times that suffocation appears imminent, also occur. Other cranial nerve nuclei may become involved, and there is always weakness of the limbs. In the most severe cases the mental faculties become blunted early, and coma develops quickly, death usually resulting within a week, though less severe cases may live long enough for affected muscles to show signs of atrophy.

Diagnosis.—Meningitis and cerebral hæmorrhage are the chief conditions from which encephalitis has to be distinguished. The absence of convulsions or spasm, and the early occurrence of mental dulness, stupor, and paralytic phenomena, make an encephalitis more probable than a meningitis; but it may be quite impossible to distinguish between the two conditions. Cases of cerebral hæmorrhage more commonly occur in older people in whom there are evidences of arterial degeneration, the onset is more sudden, and coma more profound. Polio-encephalitis is distinguished from polio-myelitis by the implication of cranial nerves, when these are affected; the normal electrical reaction of the muscles of the affected limbs; and, when present, the exaggerated condition of the tendon-jerks.

Prognosis is very variable; each case must be judged according to the severity of the symptoms. Although the prognosis is always serious, a considerable proportion of cases of acute hæmorrhagic encephalitis recover, some completely, but others with permanent mental defects, epilepsy, and paralysis with contracture, to which athetoid or choreiform movements may be added. The more sudden the onset and severe the symptoms, the worse the prognosis. The only form of superior polio-encephalitis, in which recovery may take place, is that resulting from some infectious process; but a sudden onset and a subnormal temperature are of the gravest import. The prognosis is always most serious in polio-encephalitis inferior; but even in this variety recovery has been known to result. According to Oppenheim, evidence of involvement of the white matter is of the most serious moment.

Treatment.—The same rules laid down for the treatment of meningitis must be followed in these cases. The precise course adopted must, however, depend on the state of the patient; thus antiphlogistic measures, which are admissible in the robust, are quite out of the question when those debilitated by previous disease are under treatment. It is questionable whether drugs like mercury and iodide of potassium are of any use in the acute stage, though the latter may be of service in the more chronic cases. Headache must be relieved by bromides, antipyrine, and the like; and on no account must morphine be administered. For remaining hemiplegia, massage, electricity, and cold douches, with friction, must be employed; while for the relief of epileptic fits bromides are of service.

CHRONIC ENCEPHALITIS.

We know very little of chronic inflammation of the brain, the chief difficulty being that processes degenerative in origin may lead to sclerotic changes, indistinguishable from those in which a chronic inflammation is responsible for the morbid appearances met with. Apart from sclerosis, the result of an antecedent acute inflammation of the brain, there are certain cases in which the condition appears to be chronic from its commencement.

Etiology.—Injury to the head in children has been followed by such a result. Possibly chronic alcoholism may be responsible for some cases; and, according to Gowers, a group of cases is met with in persons of gouty diathesis in which the symptoms suggest a chronic inflammation.

Symptoms.—In the present state of our knowledge, it is impossible to separate clinically, with any degree of certainty, cases in which the cerebral sclerosis is of inflammatory origin from those in which it is not. Both classes of cases are of slow progress, and may be attended by various degrees of dementia, impairment of speech, twitchings, epileptiform convulsions, and different forms of cerebral paralysis. When present, symptoms which suggest that the process is inflammatory are slight elevation of temperature, severe headache, vertigo, vomiting, and optic neuritis.

Treatment.—Where there appears to be sufficient grounds for the diagnosis, the patient must be kept as quiet as possible, and free from all mental worry. Mercury must be given in some form in the earlier stages, and its administration supplemented by measures calculated to improve the patient's general condition. Where the disease is of some standing, less good can be expected to result from the use of mercury than from drugs like arsenic and nitrate of silver, both of which are known to be antagonistic to connective tissue and neuroglia proliferation.

J. S. RISIEN RUSSELL.

CEREBRAL ABSCESS.

Cerebral abscess, or suppurative encephalitis, is a condition in which collections of pus occur in the substance of the brain, or on the surface of the organ, between the membranes and the cortex cerebri.

Etiology.—**Trauma.**—Injuries of the scalp, cranium, or brain are responsible for a large proportion of brain abscesses. The wound is commonly an open one, which readily becomes infected; injury of the soft parts may alone be responsible for abscess, the bones being quite intact, or the bones may be fractured, spicules of the inner table lacerating the brain, or caries may follow the injury. The abscess usually exists in the part of the brain immediately beneath the seat of trauma, but in rare cases it may be on the opposite side of the organ. Foreign bodies which enter the brain may carry septic matter with them and thus cause an abscess, or the wound caused by a foreign body may subsequently become infected.

Local inflammatory and other conditions.—(*a*) *Disease of the ear.*—If we except traumatic and pyæmic cases, the majority of patients suffering from cerebral abscess are the subjects of suppurative inflammation of the tympanum, a condition which is always a source of danger, owing to the frequency with which it is responsible for grave affections of the brain and its meninges. The ear mischief is as a rule chronic, and there is usually caries of the bone. Though often commencing in early childhood, in connection with an attack of one of the exanthemata, it may not lead to this serious result until well on in adult life, a fact to be borne in mind when the question of life insurance is under consideration in such cases. It is rare for acute inflammatory affections of the ear to lead to cerebral abscess. Cessation of discharge from the ear, often viewed with favour by the patient, may in reality be a serious sign, as many cases of abscess follow such an arrest of discharge. A fresh stimulus given to the ear disease, either by a blow on the ear or exposure to cold, may at any time light up a more active local process, which may be followed by abscess. The bone separating the tympanic cavity and mastoid cells from the intracranial cavity is exceedingly thin, and thus is easily destroyed, the most common points of perforation being the tegmen over the tympanum or antrum, and the sigmoid groove, in addition to which, numerous vascular, perivascular, perineural, and lymphatic channels exist, by which infective agents may reach the intracranial cavity from the tympanum, so that it is an easy matter for the cerebral meninges to become infected, with or without similar infection of the brain, or the infective material may reach the interior of the substance of the brain and produce an abscess without first setting up a meningitis. Suppurative processes of the external ear may extend to the brain by the lymphatics, leaving the middle ear intact.

(*b*) *Disease of the nose.*—Chronic suppuration in the nasal cavity and ethmoid bone is another source of cerebral abscess, which, though less common than ear disease, is of great importance. In some cases the mucous membrane of the nose is alone affected, but, more commonly, the ethmoid, sphenoid, and nasal bones are diseased, syphilis being frequently the cause of this.

(*c*) *Disease of the orbit.*—Suppuration and new growth have both been responsible for this mode of origin of abscess, but much less frequently than any of the other local causes already considered.

(*d*) *Other inflammatory processes.*—Apart from injury, caries of the bones of the skull, other than the temporal, very exceptionally gives rise to abscess. Fagge speaks of having found abscess of the brain in a case in which ulcerating epithelioma of the lip and cheek had extended to the base of the skull along the third division of the fifth nerve. Chronic ulceration about the mouth may supply the infection, and thrush of the mouth and throat has, in rare instances, caused abscesses in the brain which have been multiple and small, and have contained the oïdium albicans.

(*e*) *Cerebral tumour.*—An exceedingly rare form of local origin of cerebral abscess, is due to disintegration of tuberculous tumours in the brain.

Distant suppurative processes.—In this class of cases the infective agents are brought to the brain from distant parts of the body, by means of the blood current, and cause metastatic abscesses. This condition is met with in general pyæmia and in ulcerative endocarditis. The resulting abscesses are multiple and small, and may influence but little the manifestations of the general disease.

Of greater frequency than these is a group of cases known as the pulmonal cerebral abscess, in which condition, generally secondary to suppuration in the lungs or pleuræ, an abscess in the brain develops, without any symptoms of blood poisoning, and without infarcts or abscesses in any other organs. Bronchiectasis with fœtid secretion, abscess, or gangrene of the lung consequent on breaking down of an imperfectly resolved pneumonia, and empyema, are most frequently responsible for this form of abscess. Suppuration in some other part of the body may similarly, though much more rarely, give rise to brain abscess, in which category liver abscess must be mentioned as a source from which infection may be derived. In a case of Gibson's recorded by Cassels Brown, a solitary cerebral abscess containing staphylococci was the result of typhoid fever.

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Specific fevers.—Abscess of the brain may be a sequel to influenza and other specific fevers.

Idiopathic abscess.—In a small proportion of cases of cerebral abscess no definite cause can be found, and they have accordingly been designated idiopathic. How many of these, if any, are truly so, it is difficult to say, for there is always the possibility that slight old ear disease, or some similar mischief, has escaped recognition, or that some antecedent blow or injury to the head has been forgotten.

Morbid anatomy.—Abscesses of the brain are most commonly solitary, and vary from the size of a walnut to that of the greater part of a cerebral hemisphere. In a much smaller proportion of cases the abscesses are multiple, in which case they are usually small. They vary much as regards the amount of limitation by any definite capsule; the more acute the abscess, the less tendency is there for it to be circumscribed, and the more chronic it is, the more defined is its capsule. In the former case its limits are ill-defined, its walls shreddy, and its contents a mixture of greenish viscid pus, broken-down brain matter, and reddish débris, while in cases of longer standing the pus is bright green in colour, and it may be quite mucoid, while in the cases of longest duration there is a well-defined smooth capsule of connective tissue, which prevents the process from extending beyond its limits, and the pus thus enclosed may be quite thick and cheesy. The contents of the abscess may be odourless, may have a sickly smell, or one resembling sulphuretted hydrogen, or it may be horribly fœtid. The pus is usually acid, but in cases of long standing may be alkaline in reaction. The brain substance for a varying distance around the abscess is much softened and œdematous in appearance, a condition in part due to extension of inflammation, and in part to pressure. The increased pressure further manifests itself by flattening of the convolutions of the affected hemisphere, if the abscess is of any size.

If situated near the surface of the brain, the encephalitis may be associated with a purulent meningitis. In other cases the meningitis and encephalitis have a common origin, the former preceding the latter in point of time of development. There is, however, a singular group of cases, by no means uncommonly met with, in which an abscess situated deep in the substance of a cerebral hemisphere is separated by healthy brain tissue from the point from which the infection was derived.

The seat of an abscess depends to some extent on its cause; thus the temporo-sphenoidal lobe and cerebellum are most commonly affected in cases secondary to ear disease, the former seat being more common than the latter; the frontal lobes are usually the seat when disease in the nose supplies the source of infection, and the subjacent brain substance in the case of trauma in any region of the skull. But, apart from such considerations, abscess is more common in the cerebrum than in the cerebellum.

Pathology.—When discussing the etiology of cerebral abscess, we saw that, apart from direct infection and direct extension from inflammation in some adjacent part, infection may reach the cerebral substance through venous thrombi, arterial emboli, perivascular and perineural spaces, and lymphatic channels. This infection is in all cases dependent on micro-organisms, and the same organisms have been found both in the brain abscess and in the diseased part from which the infection was derived. Various forms of micro-organisms have been found under these conditions, but those most commonly met with are varieties of staphylococci and streptococci. No single variety of micro-organism can be held responsible for brain abscess, nor do the organisms which occasion it differ from those which induce other infective processes in the intracranial cavity, if we except the diplococcus pneumoniæ, so commonly met with in meningitis. In a case of cerebral abscess operated on by Victor Horsley, a diplococcus was isolated by Butler Harris; it caused a spreading œdema, and was pathogenetic to guinea-pigs. By its action on the blood vessels the infective agent results in œdema of the brain, with exudation of leucocytes, and, it may be also, of red blood cells; and the area of encephalitis thus produced rapidly results in disintegration of brain substance and pus formation.

The symptoms produced by an abscess in the brain depend on the local irritative effects of the infective inflammation, septic absorption, and increased intracranial pressure. Among those probably dependent on increased pressure, the subnormal temperature and slow pulse deserve some attention. There seems little doubt that the slowing of the pulse is due to increased pressure, as it is a common feature of other morbid processes accompanied by such increase of intracranial pressure, and the pulse always becomes rapid when the pressure is relieved, as by the evacuation of a cerebral abscess. With regard to the lowered temperature, the problem is not quite so simple, for it has been suggested that this may depend on the toxic effects of septic absorption; that this is not probable, however, is made clear by the fact that if the same organisms responsible for the abscess with subnormal temperature escape to the meninges, a high temperature rapidly results. It is therefore probable that the elevation of temperature which follows the evacuation of an abscess is made possible by the relief of pressure, and is not due to the removal of organisms whose toxines had been acting deleteriously on the heat-centres.

Symptoms.—It is of primary importance that we should recognise clearly that small multiple abscesses, or even single large ones, may exist in the brain, without giving any indication of their presence for a considerable period of their course, and that even when such indications do become manifest they may be of the vaguest kind possible. No greater mistake can be made than to suppose that suppuration in the brain of necessity gives rise to all the acute manifestations which are commonly met with in abscesses in other parts of the body, and sometimes in the brain itself. This especially applies to so-called idiopathic abscesses, and those coming on gradually and insidiously in connection with chronic middle ear disease, and after apparently trivial injuries of the head. So, too, any symptoms produced by the occurrence of metastatic abscesses in the brain, in the course of general pyæmia, are masked by the delirium and stupor consequent on the septicæmic condition. The opposite state of things is met with when an abscess results from an extensive injury to the head, or in any form of abscess which develops acutely and enlarges rapidly. Two classes of symptoms then show themselves, those due to the inflammatory process, and those due to the existence of a foreign body within the cranial cavity, disturbing function by pressure, irritation, and destruction of the nerve elements. The former produce general symptoms, while focal symptoms result from the latter.

Of the general symptoms produced by a cerebral abscess, pain is, as a rule, the earliest and most persistent. The headache is dull and deep-seated, and is frequently referred chiefly to the seat of the abscess, though this is by no means always the case, and it may vary in position from time to time. It also varies in intensity in different cases and at different periods of the same case.

Purposeless vomiting is another symptom of the greatest importance, and is especially frequent in cerebellar abscess. It may occur after meals, or quite apart from the taking of food. Vertigo is frequently associated with vomiting, and like it is of frequent occurrence when the cerebellum is affected; but both these symptoms may also occur when the abscess is in the cerebrum.

Optic neuritis is a most important sign, and is especially frequent in the later stages of these cases. A careful watch should therefore be kept on the discs from day to day, in any case of suspected cerebral abscess, where the indications are vague or altogether absent.

The state of the temperature is no less important an indication of the condition of things which exists within the cranial cavity than are the symptoms that have already been mentioned. It is primarily our duty to insist on the extreme importance of recognising that a subnormal temperature is not only compatible with the existence of cerebral abscess, but that it is the rule to meet with this state of the temperature in all but the earliest and latest stages of the affection, in the former of which the temperature may be slightly raised, and in the latter hyperpyrexia may result. It not uncommonly happens that a cerebral abscess is missed clinically, owing to a belief, which is widely prevalent, that suppuration in the brain cannot be present if the temperature is not raised. It is only in exceptional cases that the temperature runs an elevated, and it may be irregular course, accompanied by rigors and sweating. It is unusual for rigors to occur at any other than the initial stage of the affection, at which time they are among the most constant symptoms.

A slow pulse accompanies the depressed temperature, the rate being commonly 40 or 50, or even lower, as in a case referred to by Huguenin, in which there were only 15 or even 10 beats in the minute. The pulse is, however, increased in frequency in the earliest stage of cerebral abscess, and also when total dissolution is imminent. The respirations are slow, a feature that is more marked in cerebellar than in cerebral abscess, and, though usually regular, they may become irregular, or even Cheyne-Stokes in rhythm, more especially in the last stages of the disease.

The general health may be but little affected at first, but as a rule there is evidence of indisposition; the face becomes pale and wanting in expression, the patient loses flesh, is unable to sleep, becomes melancholy and morose, and the intellectual faculties become blunted. There is loss of appetite, the tongue becomes dry and brown, and the bowels are constipated. Towards the terminal stages of the affection there is often delirium, mental depression or enfeeblement, which conditions may pass on to stupor, coma, and death, an event which may also come about at any stage of the course of a cerebral abscess, from sudden failure of respiration, rupture of the abscess into the lateral ventricles, or some other cause.

The focal symptoms of cerebral abscess depend on the precise seat of the collection of pus, and are often absent, owing to the silent regions of the brain being so commonly affected. Alteration in character, dementia, or other mental defects may be observed early, when the frontal lobe is affected, or there may be an entire absence of any localising symptoms when this part of the brain is involved. Abscesses in the region of the sensori-motor cortex may result in limited epileptiform seizures, in paralysis of one or other limb, or the side of the face; and if the hinder part of the left inferior frontal convolution be involved, motor aphasia results. As the abscess increases in size, so the paralysis spreads, each new phase of it being possibly ushered in by further epileptiform seizures. While abscess in the temporo-sphenoidal lobe may give rise to no localising symptoms in some cases, in others a large abscess may result in pressure on the adjacent parts of the ascending frontal and parietal convolutions, resulting in paresis of the opposite side, in which the face suffers more than the arm, owing to the difference in the proximity of these centres to the seat of lesion, or if on the left side, pressure may be exerted on the posterior part of the third frontal convolution, and thus motor aphasia may result. Sensory aphasia is, however, more common in these cases, word-deafness resulting from destruction of the posterior half of the left temporo-sphenoidal convolution, and word-blindness when the adjacent angular convolution is involved. More complete hemiplegia, or hemiplegia in which the leg suffers more than the arm, and the arm than the face, may result from pressure on the internal capsule, or in the case of a large abscess in the centrum ovale. Hemianopsia results when the suppuration is in the occipital lobe of one hemisphere. When ear disease is responsible for the abscess, the facial and auditory nerves may, of course, be involved in the ear mischief; but, apart from this, the cranial nerve most commonly affected in the case of abscess of the temporo-sphenoidal lobe, is the third; ptosis, external strabismus, and a fixed dilated pupil resulting on the side of the lesion, owing to direct pressure by the enlarged temporal lobe on this nerve.

If the abscess has as its seat the cerebellum, there may either be a complete absence of all localising symptoms, as in the case of some abscesses of the temporo-sphenoidal lobe, or there may be the most typical picture, difficult to confound with the symptoms produced by abscess in any other situation. Why a collection of pus should in one case give such distinctive evidence of its presence and situation, while in another it gives rise to no such signs, it is difficult to say, unless the differences met with depend on the rate of development of the suppurative process, and the time that it allows for compensation to be established. Experimental researches on cerebellar localisation, supplemented by clinical and pathological observations in man, lead me to conclude that the following symptoms indicate the side of the cerebellum which is affected when the lesion is unilateral. Rotation of the subject about its vertical axis would, if in the direction of a right-handed screw entering an object, indicate a right-sided lesion, while if in the direction of a similar screw coming out of an object, it would indicate a left-sided lesion. Unsteadiness of gait, with a tendency to fall to the side opposite to the lesion, owing to a subjective feeling of a loss of balance, has to be carefully distinguished from a tendency to fall to the side of the lesion, owing to want of certainty of support from the lower limb on this side, or owing to the attitude resulting from curving of the trunk with its concavity to the side of the lesion, all of which conditions may be met with in unilateral lesions of the cerebellum. There is motor paresis of the limbs on the side of the lesion, especially marked in the superior extremity in man, in whom inco-ordination in the movements executed by this limb may be most pronounced. The eyes are directed away from the side of the lesion, and there is lateral nystagmus. Any rotatory or irregular characters of the nystagmus make it probable that the middle lobe of the cerebellum is involved. The neck and trunk are twisted with their concavity to the side of the lesion, and rigidity has been observed in the muscles of the limbs on this side. The knee-jerk is increased on both sides, but more so on the side of the lesion, and the other tendon-jerks on this side may also show a greater activity than those on the opposite side.

Unfortunately, in practice, we very rarely meet with cases in which the complete group of phenomena are present in the same patient; but most of them were present in a case recorded by Acland and Ballance, in which the abscess in the right lateral lobe of the cerebellum was successfully evacuated. While it is rare to meet with the whole group of phenomena in any one case, it not infrequently happens that some of the signs can be detected, but these are often overlooked, wrongly interpreted, or disregarded, owing to a want of knowledge of their real significance. A careful watch should always be kept for the slightest indication of any of the signs that have been detailed, and due weight given to them in our attempts at localisation

A further difficulty with which we have to deal clinically is the fact that, in addition to the phenomena which result from destruction of the cerebellum, others due to pressure may be added. Of these the effects on the cranial nerves are the most common; thus, as so often happens in increase of intracranial pressure, one or other, or both sixth nerves, may be affected, the facial and auditory nerves may be involved on the side of the lesion from the same cause, and there may be paralysis of the opposite limbs from pressure on the pyramid above its point of decussation. Thus affection of the facial and auditory nerves on the side of the lesion may be associated with paresis of the limbs on the same side, the result of cerebellar destruction, or they may be associated with paresis or more pronounced paralysis of the limbs on the opposite side, the result of pressure on the pyramid above its point of decussation. Except where the facial nerve is thus involved, hemiplegia, the result of a cerebellar lesion, is characterised by an absence of paralysis of the face.

At times the phenomena present are so complex that they baffle all attempts at localisation, this being largely due to the effects of increased intracranial pressure, and possibly in some measure to implication of the middle lobe of the cerebellum. Among the clinical phenomena difficult of explanation is the absence or great diminution of the knee-jerks met with in some cases of cerebellar disease; that this is in some way related to the general increase of intracranial pressure, is made probable, owing to the fact that the same condition of knee-jerks may be met with in abscesses and tumours of the cerebrum, while it never results from ablation of the cerebellum in animals. Macewen attaches considerable importance to the alteration in the character of the percussion note of the skull, which becomes more resonant when the lateral ventricles are distended, and indicates involvement of the cerebellar fossa.

Diagnosis.—The difficulties which attend diagnosis are often very great, and may even be insurmountable. The discovery of one of the recognised causes of abscess is of primary importance, suggesting the probable nature of the intracranial mischief.

With regard to acute cases, more especially those following injury, there is rarely any difficulty; rigors, early elevation followed by depression of temperature, cerebral symptoms including delirium, epileptiform convulsions, paralysis, and optic neuritis, point clearly to the probable nature of the affection. In the absence of fever the possibility of meningeal hæmorrhage has to be taken into consideration, and the difficulty may be further increased by the absence of rigors and optic neuritis, both of which symptoms would, if present, weigh in favour of abscess.

When attempting to distinguish between encephalitis without the formation of pus, and an abscess, reliance has chiefly to be placed on the occurrence of rigors, and on the length of time that symptoms have lasted. An encephalitis of septic origin is almost certain to result in the formation of an abscess, especially if there have been evidences of its existence for two weeks or longer; whereas an encephalitis of traumatic origin may continue much longer without resulting in suppuration.

In distinguishing abscess from meningitis, it must be remembered that the two conditions may coexist, and that the symptoms due to the meningitis are especially liable to obscure those the result of the abscess. The patient with acute leptomeningitis is irritable; he is extremely sensitive to all forms of external stimuli; his temperature is high and his pulse rapid—a clinical picture which differs materially from that presented by an uncomplicated case of cerebral abscess. Reliance has further to be placed on the fact that the cranial nerves are more commonly affected in meningitis, and are picked out more at random. Any focal symptoms of central origin indicate a circumscribed lesion, and thus rather point to abscess.

Infective thrombosis of the sigmoid sinus is, like the last condition, commonly present with cerebral abscess. The symptoms which characterise this affection are pain in the upper part of the posterior triangle of the neck, below the mastoid and along the jugular vein; the temperature is high, and remits markedly; there are frequent rigors, followed by sweating, and the pulse is rapid. There may be evidence of infarcts in the lungs; diarrhœa is common, and in some cases the symptoms resemble those due to enteric fever.

As chronic ear disease may, according to Gowers, be responsible for general cerebral symptoms and even optic neuritis, without the development of abscess, it can readily be understood how difficult the problem may be sometimes. Happily the number of such cases is small, so that the presence of general cerebral symptoms and any degree of optic neuritis would, if ascribed to abscess, be correct in the majority of instances.

The chief difficulty which arises in connection with chronic abscess is its distinction from intracranial tumour. The absence of any of the known causes of abscess would be in favour of tumour, as would the absence of rigors, the slow development of symptoms with remissions from time to time, the implication of cranial nerves, especially if extensive, and the presence of severe optic neuritis.

Prognosis.—The prognosis depends on the degree of certainty with which the diagnosis can be made, the presence or absence of localising symptoms, and the amount of probability there is that the surgeon will be able to successfully reach the seat of suppuration. But even when this is accomplished, the prognosis, though greatly improved, remains doubtful, in that a diffuse encephalitis or a purulent meningitis may spread from the seat of the abscess and bring about a fatal issue; or, without any such complications, it may be impossible to effect satisfactory drainage of the abscess, owing either to the character of the débris which so often has to find a means of escape, or to the fact that numerous loculi exist, so that, while some are drained, pus remains pent up in others, and thus the process of suppuration in the brain extends, and terminates fatally, in spite of a means of exit having been afforded to some of the pus. So, too, when operation is too long delayed, so that coma is pronounced, the pulse commencing to be rapid, respiration irregular, or of Cheyne-Stokes rhythm, and the temperature beginning to rise for the final hyperpyrexia which ushers in death, a fatal termination can rarely be averted.

Evidence of rupture of the abscess into the lateral ventricles justifies no other than a fatal prognosis; whereas the longer symptoms of abscess are in existence, the greater chance is there for the pus to become encapsulated, and for further spread of the inflammatory process to be thus prevented. The contents of such abscesses may become inspissated, and have been known to be latent for a great many years; in cases of the kind some hope may be held out that the abscess will not be ultimately responsible for death. The amount of hope that is thus given must, however, also depend on whether the cause of the abscess is still in existence or not, and on the possibilities there are for avoiding injuries calculated to reawaken the dormant process. In no case in which the abscess can be localised, and in which there is a reasonable chance of evacuating it, should it be allowed to remain untreated, so as to make it necessary for us to consider points in prognosis such as those last discussed.

Treatment.—Before dealing with the treatment of abscess itself, it is all-important that we should clearly recognise that in no mean proportion of cases it is a preventable disease, and that therefore suitable prophylactic measures must be adopted to suit the exigencies of each particular class of cases. The treatment of injuries and fractures concern the surgeon, and the modes of dealing with fœtid conditions of the lung, empyema, liver abscess, and the like, do not come within the scope of this article; but one condition is so important in this connection, that, although outside our immediate province, it must be dealt with briefly—this is the antecedent condition of the ears, which so frequently is responsible for the graver cerebral trouble. Ear disease, whether chronic or acute, should be always regarded as a source of danger, and accordingly no pains should be spared to promote the free escape of discharge from the ear, and to thoroughly cleanse and disinfect the seat from which the discharge is derived. Any evidence pointing to extension of the disease to the mastoid, or to the development of intracranial mischief, should be the immediate signal for trephining the mastoid, clearing out its antrum, scraping out the ear, removing all necrosed bone, and exploring the adjacent membranes and sinuses. If these measures are not followed by relief of the general symptoms of intracranial mischief, in spite of the non-existence of localising signs, the skull should be opened, and the brain explored in one or other or both of the directions in which an abscess is most commonly situated. For the exploration of the lateral sinus, temporo-sphenoidal lobe, and cerebellum, the modification of Barker's guides by Dean, in order to allow all these parts to be explored through one trephine opening, is of considerable advantage in cases in which there are no localising signs. The pin of the trephine is placed 1 in. behind and ¼ in. above the external auditory meatus, and this opening, which is partly over the lateral sinus, can be enlarged slightly upward, so as to allow the temporo-sphenoidal lobe to be explored, or downward and backward for similar exploration of the cerebellum.

Where we are fortunate enough to discover localising symptoms in cerebral or cerebellar abscess, the skull must be opened over the indicated seat of lesion.

In addition to these surgical procedures, by which we can alone hope to cure the affection, much can be done to lessen the tendency of the inflammatory process to spread, by keeping the patient at rest, avoiding exposure to cold, blows to the head, and mental excitement. Cold to the head and local depletion by means of leeches are of distinct advantage, and the patient's general condition should be kept up by means of a tonic line of treatment.

Antipyrine and similar remedies should, as far as possible, be given preference over narcotics for the relief of pain, in that the latter are so apt to mask symptoms which might otherwise give us some indication of the probable seat of the suppuration within the cranial cavity; but it is, of course, not always possible to avoid the use of sedatives where pain is severe.

J. RISIEN RUSSELL.

HYPERÆMIA.

Cerebral hyperæmia, active or passive, is due to overfilled capillaries; the former condition is caused by increased arterial flow, the latter by obstruction to the outflow of blood from the venous sinuses.

Arterial hyperæmia of the brain produces no results of importance; but cerebral venous congestion is of great pathological importance, because the blood in the capillaries is deficient in oxygen and contains excess of carbonic acid. The cranium being a closed cavity, a rise of the general venous pressure, interfering with the outflow of venous blood, prevents arterial blood from entering the brain in sufficient quantity, although some compensation occurs by the displacement of the cerebro-spinal fluid from the subarachnoid space into the spinal canal. The amount of blood in the brain, however, varies comparatively little in quantity, and if there is an increase of venous blood within the cranial cavity, there is almost a proportional decrease of arterial. Leonard Hill, who has made a very careful study of this subject experimentally, believes that there is little evidence to show the existence of cerebral vasomotor nerves; he considers that the cerebral circulation is controlled mainly by the action of the vasomotor centre acting on the splanchnic area and by the mechanism of respiration. The vasomotor centre acts as a self-adjusting mechanism; for anæmia, whether due to deficiency of blood in the cerebral capillaries or the anæmia of passive congestion, excites the vasomotor centre, causing constriction of the splanchnic arteries, a general rise of blood pressure, and an increased flow of blood through the cerebral capillaries.

Etiology.—The causes of active congestion are—Increased action of the heart, especially when due to excitement, unaccompanied by mechanical obstruction in front; chill of the surface of the body, causing constriction of the superficial vessels; suppression of the menses; constipation; and various toxic substances, *e.g.* nitrite of amyl, nitroglycerin, and alcohol. The last-mentioned may, however, act by quickening the heart's action. In persons past middle life there is a condition termed plethora, the symptoms of which are headache, giddiness, flushing, following exercise and meals: and these are, on insufficient grounds, attributed to hyperæmia of the brain.

The causes of passive congestion are mechanical, and due to those conditions which lead to overfilling of the venous system generally, or which impede the outflow of blood from the cerebral sinuses, *e.g.* heart disease, lung diseases (such as chronic bronchitis, emphysema, whooping cough), status epilepticus, tumours pressing upon the superior vena cava, innominate veins or veins in the neck, pressure upon or thrombosis of one of the cerebral sinuses. Finally, in all conditions which lead to death from suffocation, passive congestion occurs in its most intense form.

Morbid anatomy.—The appearance of the vessels of the brain after death depends so much upon the mode of death, and the relative position of the head to the body subsequently, that it is difficult to distinguish the essential from the accidental conditions. A great deal too much importance has been attributed to the appearance of the blood vessels of the brain post-mortem, and many symptoms during life have been attributed to the congested vessels; but, seeing that these same appearances of congestion may exist in persons who have died without any symptoms, little value can be attached to them.

Active congestion disappears after death, and when distension of the minute vessels is found post-mortem without marked engorgement of the veins, it indicates inflammation rather than congestion. Still, in prolonged passive congestion, the veins and sinuses may be found engorged with dark blood, and the grey matter dark in appearance, owing to distended capillaries; and in extreme cases even minute hæmorrhages may exist. The writer has examined microscopically the brain in numerous cases of congestion from status epilepticus. Not only are the veins congested, but the perivascular and perineuronal spaces are dilated, and chromolytic changes are observable by Nissl's method in the large pyramidal cells.

Symptoms.—Active congestion may be attended by general irritation, combined with feelings of heat in the head, redness of the face, throbbing carotids, headache, vertigo, noises in the ears, and black spots before the eyes. It is especially liable to occur in people who are neurotic, neurasthenic, or hysterical, and the exciting cause may be some psychical disturbance. Formerly, almost all forms of convulsions were ascribed to cerebral congestion, but, according to Gowers, simple convulsions from this cause are extremely rare.

Venesection was practised formerly for plethora, to relieve cerebral congestion, and thereby remove headache, giddiness, and mental irritability. The writer agrees with Leonard Hill in considering that arterial hyperæmia may be the sequence and not the cause of disturbance of the central nervous system.

Passive congestion is often attended by frontal headache, aggravated by coughing, confusion, and a sense of fulness of the head, but there are no characteristic or constant features of this condition; and, as an independent clinical entity, cerebral hyperæmia probably does not exist. When there is much cyanosis, there may be dulness of the intellect, drowsiness, torpor, and even coma, due to anoxæmia and excess of carbonic acid in the blood.

Treatment.—In passive congestion gravity plays an important part, consequently the upper part of the body should be raised; hot foot-baths and sinapisms used to drive the blood from the head, and purging to relieve the portal congestion. Ice-bags may be applied to the head, and in some cases venesection may be resorted to, in order to relieve the distended right heart and overfull venous system. In plethoric people it is essential to regulate the diet.

F. W. MOTT.

ANÆMIA.

Cerebral anæmia may be temporary and sudden, as in syncope, which is due to failure of the blood supply to the brain, owing to sudden inhibition of the vasomotor centre, whereby dilatation of the splanchnic vessels occurs, and when the heart, which may be inhibited also, is unable to maintain the column of blood in the cerebral vessels against gravity; the result is cerebral anæmia and temporary loss of consciousness. The immediate causes are generally of a reflex nature, *e.g.* psychical influences, sudden fright or emotion at the sight of blood or wounds, etc.; oppressive heat, gastric disturbances, blows or injuries of the abdomen, evacuation of large quantities of fluid from the abdomen or pleural cavity, and various forms of heart disease. Especially are any of these causes likely to cause a fainting fit, if the patient is delicate, anæmic, or convalescing from a severe illness.

Pathology.—The anatomical condition of the brain in anæmia is characterised by pallor of the membranes; the large veins and sinuses alone contain blood. The small veins over the convolutions are empty, and there is an excess of cerebro-spinal fluid; the grey matter looks extremely pale, and there are but few puncta cruenta. The fact that anæmia produces convulsions affords evidence of an increased irritability on the part of the motor neurons. Experiments on animals show that both carotids and both vertebral arteries can be tied, and, provided collateral circulation be restored within a short time, all the symptoms of the anæmia, such as paresis, dementia, and blindness, pass off. Examination of the brains of these animals by Nissl's method shows very little change in the nerve cells of the cortex. Likewise, the writer has found in extreme cases of anæmia very little change in the normal appearances, and unless the supply of blood to the brain is cut off entirely for some hours, no important permanent changes occur in the nerve cells. In animals in which the collateral circulation is not restored soon enough, the nerve cells undergo marked changes, which end in their complete dissolution.

Symptoms.—Swimming of the head, buzzing in the ears, black spots before the eyes, objects moving round, a feeling that the earth is sinking under the feet, and nausea which frequently terminates in vomiting. If the patient can be placed in a horizontal position soon enough, that is before loss of consciousness occurs, the blood supply to the brain is restored, and the attack may pass off. If not, the loss of consciousness may last from a minute to half an hour; one of the most marked features is pallor and a cold sweat, with a small, frequent pulse. Usually such an attack of syncope is not dangerous, except it be due to heart disease. Anæmia of the brain may arise from sudden loss of blood, and a condition of syncope occur, such as above described; but if the amount lost is very great, as in wounds of large arteries, post-partum hæmorrhage, etc., general convulsions of an epileptiform character may occur; the pupils dilate, the reflexes are abolished, and there is complete loss of consciousness.

The usual phenomena met with in continuous cerebral anæmia, such as occurs in diseases attended by frequent hæmorrhages, and the various forms of impoverished blood diseases, such as chlorosis, pernicious anæmia and leucocythæmia, are headache, sleepiness, buzzing in the ears, swimming of the head, feeling of sickness, and dimness of vision. Convulsions are rare, but there is frequently muscular weakness. Seeing that gravity plays such an important part in the cerebral circulation, all these phenomena are naturally increased by the erect posture, particularly when it is suddenly assumed. Ligature or compression of a carotid artery may produce transient weakness and numbness of the opposite side of the body, and if the cerebral vessels are diseased, convulsions and paralysis even may ensue. Fagge mentions that he had notes of five cases of ligation of the carotid, producing symptoms. A condition termed by Marshall Hall hydrencephaloid, because the symptoms resemble those of acute hydrocephalus, sometimes occurs in young children suffering from very severe diarrhœa. The symptoms have been referred to cerebral anæmia, and are as follows:—A somnolent drowsy state, pallor of the face, contracted pupils, and depressed fontanelle, with occasionally strabismus and rigidity of the neck. In fatal cases death is preceded by dilatation of the pupils and coma.

Prognosis and treatment.—The prognosis of cerebral anæmia depends upon the cause, and is, as a rule, favourable when there is no organic disease of the heart vessels or brain. Treatment depends entirely upon the cause, and is based upon the following general principles:—(*a*) Improving the condition of the blood and the general nutrition of the body; (*b*) affording immediate relief of the symptoms by placing the patient in the horizontal position, and thus removing the influence of gravity; (*c*) by stimulants and cardiac tonics, to increase the heart's action; and (*d*) to afford support by applying a firm flannel bandage around the abdomen.

F. W. MOTT.

CEREBRAL HÆMORRHAGE.

Syn., Fr., *Hémorrhagie cérébrale;* Ger., *Gehirnblutung;*
Lat., *Hæmorrhagia Cerebri; Apoplexia Sanguinea.*

Etiology.—The essential cause of cerebral hæmorrhage is disease of the vessel walls; it is generally, but not necessarily, associated with an increase of the arterial pressure. All those pathological conditions which lead to disease of the vessel wall may be factors in the production of apoplexy.

The most important and frequent cause is a disease of the small arteries, which leads to the formation of miliary aneurysms. Charcot and Bouchard in 1868 showed that in seventy-seven consecutive cases of cerebral hæmorrhage, minute aneurysms, varying in size from $\frac{1}{100}$ to $\frac{1}{25}$ in., existed upon the arterioles of the brain; they were very numerous, as many as one hundred existing in one brain. They are round or spindle-shaped, and are caused by degenerative changes of the intima, associated with degeneration and atrophy of the muscular fibres of the media. This degeneration is only a portion of the universal arterio-capillary degeneration described by Gull and Sutton as arterio-capillary fibrosis (arterio-sclerosis). There is in most cases associated cardiac hypertrophy, granular contracted kidneys, and atheroma; and not infrequently emphysema. Hæmorrhage from this cause seldom occurs in persons under 40; it is more frequent in men than in women, nearly in the proportion of two to one; this disproportion arises, not merely from difference in sex, but difference in occupation and habits, and the greater liability to syphilis, gout, alcoholism, and excess of nitrogenous diet in men, which as we know are important factors in the production of arterial degeneration and chronic Bright's disease. Heredity, however, apart from these factors, is a most important cause. I have observed families in whom apoplexy for many generations was unknown; whereas in other families it was the cause of death in many members. It is usually considered that people with a short, thick neck, round florid countenance, and square build of the chest, are more liable to apoplexy than others.

Of 1600 post-mortems at Charing Cross Hospital, of both sexes and of all ages, sixty died from cerebral hæmorrhage; of these, seven were due to rupture of an aneurysm, thirty occurred in subjects suffering from granular contracted kidneys and hypertrophy of the heart. The frequent association of cardiac hypertrophy points to high arterial pressure as an important factor in causing rupture, and also in the production of miliary aneurysms. The miliary aneurysms are found with relative frequency in precisely those situations where hæmorrhage is most frequently met with. Charcot places them in the following order:—Central ganglia, cortex, pons, cerebellum, centrum ovale, middle cerebellar peduncle, crus cerebri, and, lastly, medulla oblongata. Atheroma and miliary aneurysms are frequently co-existent, but there is probably not a causal connection. I have occasionally found cerebral hæmorrhage in senile dementia, and after carefully washing the vessels in running water, discovered no miliary aneurysms, although the arteries were very atheromatous. Although subdural and subarachnoid hæmorrhage is common in the post-mortem room of asylums, and the existence of atheromatous arteries is the rule rather than the exception, yet intracerebral hæmorrhage is, relatively to hospital experience, infrequent. The cause, no doubt, lies in the fact that hypertrophied heart is rare, and this is curious, seeing that a slight or moderate degree of interstitial nephritis is very common.

The exciting causes of cerebral hæmorrhage may be muscular exertion, straining at stool, emotions, coitus, psychical excitement, and cold baths. It may occur during sleep; but it must be remembered that the arterioles then are in a state of contraction, and the heart is not acting against gravity, and therefore the pressure in the diseased vessels may be even greater than usual.

Cerebral hæmorrhage may occur from rupture of an aneurysm at the base or on the surface of the brain. A not infrequent cause of cerebral aneurysm is infective embolism, either from ulcerative endocarditis or infective arteritis; this is especially met with in young rheumatic subjects. The writer has heard of cerebral hæmorrhage occurring in a young healthy woman, in whose brain at the post-mortem no aneurysm or diseased vessel was found. In children, meningeal hæmorrhage may occur; and a frequent cause of birth palsies is a rupture of a vessel during parturition. Hæmorrhage may occur in tumour cerebri; and subdural and subarachnoid hæmorrhage is frequently seen in general paralysis of the insane and senile dementia. Lastly, a number of general blood diseases may be complicated by hæmorrhage, *e.g.* leukæmia, anæmia, scurvy, and purpura; sometimes it occurs in severe cases of infectious diseases, *e.g.* smallpox and typhoid fever. The escape of blood is generally from the capillaries, and due to fatty degeneration of their walls.

Primary ventricular hæmorrhage occurs in some rare instances; it is due usually to rupture of vessels of the choroid plexus, or of the velum interpositum; the conditions are the same as those which predispose to hæmorrhage elsewhere, *e.g.* miliary aneurysms, blood conditions, or even mechanical congestion, as in asphyxia.

Morbid anatomy.—On opening the cranium, very frequently the dura mater can be seen distended, and the falx pushed over to one side, indicating a large effusion of blood, and when the surface of the hemisphere is exposed the convolutions appear flattened, and the sulci partly obliterated. This condition is caused by the pressure of the brain against the cranium. If the brain be sliced up, the extent and seat of the hæmorrhage can be ascertained; in some respects these facts can be determined better if the brain is first put into formalin solution. We have already said that the most frequent seat of miliary aneurysms is also the most frequent seat of hæmorrhage. At the base of the brain, and at the commencement of the middle cerebral artery, a number of small terminal arteries, *lenticulo-striate*, pass up through the anterior and posterior perforated spots to supply the basal ganglia, *i.e.* optic thalamus, nucleus caudatus, nucleus lenticularis, and the adjacent important tracts of fibres which form respectively the internal and external capsules (Fig. 18). It is in the neighbourhood of the external capsule that hæmorrhage most frequently occurs, and it can be easily understood how a comparatively small hæmorrhage in this region can produce paralytic symptoms; because we have in the adjacent internal capsule all the afferent and efferent projection systems of fibres which convey motor and sensory impulses. There is one artery in particular, situated outside the left lenticular nucleus, which, from its frequent rupture has been designated the artery of hæmorrhage (Charcot). According to Gowers, it occurs with equal frequency in both hemispheres. When the hæmorrhage is great, it tears up the brain substance, and blood may then enter the lateral ventricle, and in severe cases all the ventricles may be filled with blood; it may even escape into the subarachnoid space at the base of the brain. Sometimes, but rarely, it may tear up the centrum ovale and pass through the cortex to the surface of the brain. In subpial hæmorrhage it is frequently a vein that has given way, the blood fills up the adjacent sulci and fissures, and the convolutions are often eroded but not greatly disintegrated.

If the clot and vessels be placed in a basin of water, into which a gentle stream of water is allowed to run so as to wash away all the blood and attached brain substance, small globular or spindle-shaped swellings can be seen on the smallest arterial twigs. The largest are about the size of a pin's head, and are often dark in colour, owing to the contained blood clot; these are the miliary aneurysms.

In recent cases the effused blood is dark in colour, generally clotted, and often mingled with lacerated brain substance. As changes take place in the clot, the colour alters first to a chocolate-brown, and later a brownish yellow. The hæmorrhage is usually single, but when small there may be more than one; sometimes evidence of a previous hæmorrhage is found in another region, in the form of a cyst, with orange-yellow staining of its walls and adjacent brain substance; or there may be a scar of connective tissue. Both conditions indicate resorption of the degenerated nervous tissue and effused blood.

Microscopical examination reveals blood and degenerated nervous tissue, fibres with their myelin sheath breaking up, granulation corpuscles, altered ganglion cells and phagocytes containing the products of disintegration. If the hæmorrhage is not quite recent, hæmatoidin crystals may be found.

Symptoms.—The apoplectic seizure "stroke" may be preceded by certain premonitory symptoms, *e.g.* giddiness, headache, fulness of the head, dimness of vision, numbness, weakness and tingling in the limbs, slight speech disturbances and mental confusion; in most cases the patient is suddenly attacked while in apparent good health. The premonitory symptoms may be explained by small hæmorrhages, or circulatory disturbances due to the co-existent atheroma.

Two factors take part in the production of symptoms, namely—(1) sudden increase of intracranial pressure; (2) laceration and destruction of brain substance. An effect of a large hæmorrhage is an increase of pressure upon the whole brain, causing coma, which may prove rapidly fatal, or pass off as soon as adjustment of pressure takes place, whereas laceration and destruction of brain substance usually lead to impairment or loss of function, the most common being hemiplegia and aphasia. The more rapidly the blood is poured out of the ruptured vessel, the more sudden is the loss of consciousness, and the greater the depth of coma. If, on the other hand, the vessel is small (unless it be in the pons or medulla), and the hæmorrhage slight, the apoplexy may only be attended by a feeling of vertigo and slight disturbance of consciousness.

Ingravescent apoplexy is a term applied to slow but progressive cerebral hæmorrhage; the patient first suffers from mental confusion, delirium, and hemiparesis, then gradually the paralysis increases and he loses consciousness. There may be all stages between a sudden and gradual onset of apoplexy; but the typical onset and symptoms will now be described. As a rule, when the medical man is called to a case of apoplexy, the patient is insensible; in the majority of instances, the history of the actual onset is not forthcoming; the patient has been found in the street insensible, lying on the floor, or in the water-closet, not infrequently the attack occurs during sleep. In rapidly fatal cases the patient falls down unconscious; probably the absolutely sudden loss of consciousness is not so common as is generally supposed; much more frequently there are premonitory symptoms, which may last from a few minutes to a few hours; then the patient sinks to the ground or into a chair and becomes insensible. The degree of unconsciousness varies; sometimes the patient is only confused, sometimes he can only be roused with difficulty; but it is very rarely that consciousness is entirely unimpaired. The writer was once called at a ball to see an elderly lady who had been suddenly taken ill. She was standing against the wall, speechless, although she was conscious, for she tried to make him understand. Occasional twitchings of the right side of the face occurred; the pulse was one of high arterial pressure; her countenance flushed, and a minute or two later she sank to the ground unconscious; general convulsions occurred, and then deep coma, which terminated fatally in a few hours from the onset. Had the writer arrived two minutes later he would have thought the onset absolutely sudden.

The loss of consciousness deepens to coma; in most cases the face is flushed generally, but there may be pallor; the pulse is full and tense, and often slowed by the increased intracranial pressure. The breathing is stertorous, sometimes sighing and slow, sometimes Cheyne-Stokes in character. The cheeks and lips are drawn in with inspiration and blown out with expiration. The muscles are relaxed and flaccid; there is escape of the urine and fæces, and the reflexes are abolished, not only in the limbs, but in the conjunctiva and iris. The attack may commence with epileptiform convulsions, and there may be conjugate deviation of the head and eyes, due either to irritation or paralysis.

The pupils may be normal in size, equal or unequal, sometimes both contracted or both dilated, or one contracted or one dilated. It is not always easy to determine which side of the body is paralysed, but observation of the face will show drawing over the angle of the mouth on one side, and the cheek on the opposite side will be seen to blow out more during expiration; again, the limbs of the paralysed side will drop more heavily when let fall, owing to the loss of tonus in the muscles; the reflexes on that side are quite abolished, whereas sometimes they can be obtained on the other side to some extent; occasionally spontaneous movements of the limbs of the non-paralysed side occur. Early rigidity of the extremities is met with sometimes, and generally is an unfavourable symptom, as it is especially liable to occur when the blood has burst into the lateral ventricle.

The temperature at first falls, and in fatal cases it may continue to fall until death; after twenty-four hours it may return to normal and remain so; or it may rise a few degrees, owing to the constitutional disturbance occasioned by inflammatory changes about the hæmorrhage; and death may take place, or, if consciousness has been regained, delirium may set in, or the patient may again become comatose. In cases of hæmorrhage into the pons or medulla, there may be an initial rise of temperature to 104° to 106°, or even more. The urine and fæces are usually passed involuntarily; if, however, it should be necessary to draw off the urine, the opportunity should be taken of testing it for albumin and sugar; the presence of the former is usually but not necessarily indicative of chronic Bright's disease, and the latter points to a lesion of the pons or medulla. In a great number of cases, the patient never emerges from the apoplectic state; the breathing becoming quickened and irregular, sometimes Cheyne-Stokes in character; mucus accumulates in the trachea and larynx; the pulse is more frequent, the eyes dim; and death, frequently preceded by a considerable rise of temperature, occurs in from two hours to two days after the onset. Acute bedsores over the buttock of the paralysed side may occur, also hypostatic pneumonia, both symptoms of grave significance. More often the patient recovers consciousness in a few hours to a few days after the onset, and the opportunity then presents itself of ascertaining the probable seat and extent of the lesion by the loss or impairment of function produced.

Diagnosis.—Probably no case can present more difficulties to a medical man than one in which he is called to see a patient who has been found in a state of coma, for not only is the diagnosis of the greatest importance in the line of treatment to be adopted, and the prognosis to be given, but serious medico-legal questions may arise. Coma may be primary, coming on as it were abruptly and without any antecedent illness of a definite nature; or secondary, occurring in the course of some disease which is known to be liable to such an association.

The causes of primary coma are: injury, causing concussion, compression, or laceration of the brain; alcoholic intoxication; narcotic poisons, sunstroke or prolonged exposure to cold; cerebral hæmorrhage; cerebral embolism; cerebral thrombosis. Before a diagnosis of cerebral hæmorrhage can be made, there are therefore certain points to be ascertained, namely, Is the coma primary or secondary to some disease which is known to be liable to the association of such a condition? The first thing to be done is to examine the head, and if there is an injury, to determine whether this is likely to be the cause of the coma—(*a*) by shock or concussion, (*b*) by damage to the skull or its contents. In such a case we may have to do with fracture of the skull or compression, meningeal hæmorrhage, or hæmorrhage into the substance of the brain. Any of these conditions may happen to a man who falls down suddenly insensible, as in an epileptic fit, but of more interest from a medico-legal aspect are those cases which have been recorded, in which a man has struck another in a quarrel, producing a head injury; the man has died from intracerebral hæmorrhage. Is it a case of accidental death, manslaughter, or murder? The wound in the head has possibly had no more to do with the hæmorrhage than the excitement of the quarrel. The intracerebral seat of the hæmorrhage, with the existence of those conditions which predispose to apoplexy, should, if present, lead to the acquittal of the prisoner.

"Drunk or dying" is too frequently the announcement in the papers of the unfortunate result of neglecting a rule that should never be departed from, namely, that a man who is insensible and who cannot be roused, even although he smells strongly of liquor, should under no circumstances be sent away to the police station, or left to himself to sleep off his supposed drunkenness (Fagge). Even supposing he is only drunk, if a more serious condition is suspected, little harm can be done by suspending judgment; on the contrary, if under the obscuring effect of intoxication a serious and fatal disease is overlooked, a grave error will have been committed.

Narcotic poisons.—Opium, morphine, chloral, chloroform, and sometimes prussic acid or cyanides, when not immediately fatal, may give rise to coma. There is generally a history of the poison having been taken deliberately or by misadventure. Sometimes a letter is left, a bottle found at hand, or the smell of the breath, to help the practitioner; failing this, even under certain circumstances, poison might be suspected, and since every minute is precious, no case requires more decision and sound judgment than such a one; because, to apply the stomach pump or give an emetic in a case of apoplexy would be highly injurious; whereas to leave a case of narcotic poisoning unrelieved would be worse. There is, however, only one form of cerebral hæmorrhage which resembles and has been mistaken for opium poisoning, namely, hæmorrhage into the pons, in which there is deep coma and pin-point pupils; it is this latter symptom which leads to the mistake; but careful observation will always reveal some other signs, *e.g.* asymmetry about the face, slight rigidity and twitchings of the limbs on one side, conjugate deviation; symptoms which are not likely to be met with in opium poisoning.

Sunstroke, heat-stroke, prolonged exposure to cold, may give rise to coma, and sometimes sunstroke may be associated with cerebral hæmorrhage. The three common causes of insensibility, or coma from organic brain disease are hæmorrhage, embolism, and thrombosis; and, after excluding other possible causes of the apoplectic condition, they should be if possible diagnosed one from another.

The causes of secondary coma are epilepsy, meningitis, especially when tuberculous, acute yellow atrophy, tumour cerebri with hæmorrhage, cerebral abscess with rupture, syncope in heart disease, diabetes, uræmia, and general paralysis. It is possible to mistake a secondary coma for the coma of cerebral hæmorrhage; but symptoms pointing to pre-existing organic disease, characteristic of the particular affection, are usually not wanting. Sometimes, however, the rupture of a latent cerebral abscess, the coma of uræmia, and the epileptiform or pseudo-apoplectiform attacks in general paralysis may be mistaken for cerebral hæmorrhage or softening. The reader is referred to the chapters upon these subjects for further particulars.

Prognosis.—The apoplectic stage.—The prognosis will depend upon evidence pointing to the extent and seat of the hæmorrhage. The intensity and duration of the coma is the most important evidence in the early stages. If the patient remains absolutely comatose for more than twenty-four hours, there is not much probability of recovery. A rapid fall and a rapid rise of temperature, associated with symptoms of bilateral paralysis in the limbs, may be looked upon as indicating a large hæmorrhage in the hemisphere, with rupture into the lateral ventricles, or a hæmorrhage into the pons, and therefore such symptoms are of fatal omen. Acute bedsores, and gradually deepening coma, are very grave symptoms; likewise conjugate deviation of the head and eyes, Cheyne-Stokes breathing, and glycosuria.

Paralytic stage.—The prognosis depends upon whether the paralysis is due to actual destruction of the cortico-pyramidal system, or to functional disturbances by pressure, anæmia, etc. If due to destruction, there must be permanent impairment or loss of function, according to the extent of the destruction. Those parts which show some return of movement within a few weeks of the attack, will recover probably useful power; whereas paralysis that remains complete at the end of a few months will probably remain considerable in degree for the rest of life. Persons who have had one attack of apoplexy are always liable to a second. If a hæmorrhage has occurred in one hemisphere, and the patient recovers, hæmorrhage into the opposite hemisphere is nearly always fatal.

Treatment.—Persons who are known to suffer from arterio-sclerosis and high arterial pressure, or whose relations have been known to have died of apoplexy, should, without unnecessarily alarming them, be advised to live a tranquil life and avoid excesses of all kinds, especially violent exertion, straining at stool, and other exciting causes of apoplexy. If the patient has already had a "stroke," these precautions are rendered doubly necessary. When called to a case of apoplexy, the medical man has first to decide the cause; but in all cases of doubt, a man who is past middle life, lying in a comatose condition, should be kept at rest. He should be laid down on his back with the head and shoulders propped up, thus favouring by gravity the flow of blood from the head. Movement should be avoided as much as possible, either active or passive, for nothing favours the arrest of hæmorrhage so much as absolute rest. Venesection, so frequently practised by our ancestors, is seldom resorted to now, although occasionally it might be beneficial when the heart's action is powerful, the arterial pressure high, and the face turgid. Ten ounces might in such a case be taken from the arm.

Free purgation by calomel or croton-oil (mixed with a little butter and placed upon the tongue) has a very powerful influence upon the cerebral circulation, by opening the splanchnic floodgates and filling the abdominal vessels with blood, and thus drawing it away from the brain. Ice-bags may be applied to the head; if the treatment does not do the patient much good, it does no harm and will satisfy the friends. Sinapisms to the back of the neck may be employed, to produce reflex contraction of the arterioles. Alcohol and stimulants should be avoided. If there is great heart weakness, sal-volatile may be given.

When the patient has recovered consciousness, his diet should for some days be only liquids. If there is sleeplessness, bromides, chloral, sulphonal, or trional may be used. The danger of bedsores should always be borne in mind and obviated as far as possible, by placing the patient as soon as possible upon a water-bed, and instructing the nurse to take great care to keep the patient clean; the position of the limbs should be changed from time to time, so as to prevent long-continued pressure on the same spot. Hot-water bottles, if applied to the feet, should be covered with flannel, and the buttocks and trochanter on the paralysed side may be daily mopped over with spirit lotion to harden the skin. The bowels should be opened by purgatives or by enema.

F. W. MOTT.

ANEURYSM OF THE CEREBRAL ARTERIES.

The subject of miliary aneurysm and its relation to cerebral hæmorrhage has already been alluded to; but aneurysm of the cerebral arteries, although usually not diagnosed until rupture has occurred, may give rise to recognisable symptoms during life.

Etiology.—Aneurysm is more common in males than females; it may exist at any period of life, but it is more frequent in the first half. The causes are the same as aneurysm elsewhere, namely, syphilitic arteritis and atheroma; but besides them and even more important is infective endocarditis, leading to embolism.

Pathology.—Aneurysm of the cerebral arteries is due to the same causes as elsewhere, namely, weakening of the muscular and elastic coats, by which a saccular dilatation or general aneurysmal dilatation of the vessel occurs. It nearly always affects the arteries of the base of the brain, and the size varies from a pea to a pigeon's egg. The arteries of the left side are more often affected than the right, the carotid system more often than the vertebral. Rupture occurs in rather more than half the cases; the blood most frequently escapes into the membranes, into the base of the brain, sometimes into the brain substance or the ventricles.

Symptoms.—Up to the time of bursting, the aneurysm may have occasioned no definite symptoms, and sudden death or ingravescent apoplexy may be the first indication of its existence. If the aneurysm is of good size, there are usually general and local symptoms.

General symptoms.—Headache usually continuous, sometimes throbbing in character, and no relation of pain to the seat of the aneurysm: giddiness, vomiting may occur, and rarely convulsions; mental confusion, dulness, or maniacal excitement and irritability. Sometimes a murmur can be heard upon auscultation; this is not, however, absolutely diagnostic, because a tumour in the neighbourhood of a large vessel may give the same. The murmur is not always heard by the patient.

Local symptoms are dependent upon the seat of the aneurysm. Aneurysm of the internal carotid may produce pressure upon one of the optic nerves, the nerves of the ocular muscles, the olfactory, and the first division of the fifth. The result will be blindness, optic neuritis or atrophy, ocular paralysis, anosmia, pain, hyperæsthesia, anæsthesia of the part supplied by the first division of the fifth; perhaps hemiparesis, from pressure on the crus cerebri. If the middle cerebral is affected, and of all arteries this is the most likely, there may be compression of the oculomotor nerve and of the olfactory, associated with motor-irritative phenomena, convulsions, hemiparesis, facial monoplegia, or aphasia.

The posterior communicating is close to the third nerve and the optic tract, and aneurysm of this vessel may produce ocular paralysis, external strabismus, and hemianopsy. The basilar artery is most frequently affected after the middle cerebral; it is usually attended with severe headache, stiffness of the neck, paresis and paralysis of the extremities; sometimes alternate paralysis, or some phenomenon pointing to irritation or paralysis of any of the neighbouring cranial nerves. Aneurysm of the vertebral may likewise cause paralysis of the extremities and involvement of the cranial nerves in the neighbourhood.

Diagnosis and prognosis.—The diagnosis is as a rule impossible; it may be surmised when there is headache with ocular paralysis on one side, coming on after some transient symptoms of embolism in a young person suffering with endocarditis. The prognosis is bad, and death usually occurs in the course of a few months or years, but sometimes spontaneous cure takes place. I have recently seen an aneurysm of the basilar as large as a filbert quite cured and filled with a dense fibrous mass. It affected one side of the artery only, and the lumen of the vessel was nearly as large as normal.

Treatment.—Little can be done. Iodide of potassium may be administered. The patient should be kept at rest, bodily and mentally, sleep should be obtained by narcotics, and pain relieved. Ligature of the carotid might perhaps be tried; but, owing to the free anastomosis of the circle of Willis, it would probably not have much effect.

F. W. MOTT.

CEREBRAL SOFTENING.

Syn., Fr., *Ramollissement cérébrale;* Ger., *Hirnerweichung;*
Lat., *Encephalomalacia.*

This is necrobiosis of an area of brain substance, occasioned by obliteration of its vascular supply. Cerebral softening was first described by Rostan in 1830; and Virchow, in 1847, pointed out that in most cases it arose either from embolism or thrombosis.

Etiology and pathology.—This disease is probably the most frequent of all organic brain diseases; it is very often met with in workhouse infirmaries and asylums, owing to the frequency of senile atheroma and the consequent chronic brain disease. Any condition which may lead to obliteration of the circulation in the cerebral vessels is liable to cause softening, for they may be blocked suddenly by embolism or by clotting of the blood within a diseased artery; although occasionally coagulation may occur in arteries or veins, independently of a vascular lesion.

Embolism.—Embolism is more frequently met with in people under 40, the subjects of endocarditis. Particles from a diseased mitral or aortic valve may be swept into the blood stream and carried to the brain; it does not necessarily follow, however, in valvular disease, that the embolus came from the vegetations on the diseased valve; it may arise from a clot in the auricle or ventricle which has undergone softening, and broken up, the débris being carried into the circulation.

Sometimes, but rarely, fragments from an atheromatous ulcer of the aorta, or from the clot of an aneurysm, may become detached and give rise to embolism. It is a matter of considerable importance whether the detached fragment carries infective organisms, and we have already alluded to the fact that ulcerative endocarditis can lead to cerebral hæmorrhage. The embolus infects the wall of the artery where it is lodged, and leads to septic inflammation and softening, with the formation of an aneurysm which may rupture. Septic organisms in uterine phlebitis may pass through the capillaries of the lungs, or the blood current may be infected by gangrene of the lungs. The capillaries of the brain may thus be obstructed by micro-organisms, which cause minute areas of suppuration, and, by their coalescence, cerebral abscess. If we exclude these microbic emboli, the source of embolic particles must be in the circulation between the pulmonary veins and the arteries of the neck.

Thrombosis.—Coagulation of blood may occur either in the arteries or the veins; the latter owing to the fact that they are seldom diseased, and their communications with one another, more numerous, much more rarely become obstructed in such a way as to produce softening. Coagulation in a vessel is favoured by the following conditions:—A rough state of the inner wall of the vessel, altered condition of the blood, and slowing of the circulation. In atheroma all these conditions exist; the vessel wall is thickened and diseased by its loss of elasticity; the circulation is slowed, and there is generally an impoverished condition of the blood, and often a weak acting heart, especially in old people. Atheroma is more likely to occur in the subjects of chronic Bright's disease, alcoholism, and syphilis; it affects especially the arteries of the circle of Willis and the large branches arising from it.

Syphilitic endarteritis is a very frequent cause of thrombosis and softening. Spontaneous coagulation is said to occur from altered conditions of the blood and a weak acting heart, independent of vascular disease. It is therefore met with in the cachexia of certain grave diseases, namely, in phthisis and cancer, in typhus and pneumonia, after parturition, and in marasmus at all periods of life, but especially in the very young and very old.

Morbid anatomy.—The arteries of the brain are terminal arteries, especially the lenticular arteries supplying the basal ganglia and internal capsule, and inasmuch as the anastomosis of these arteries with one another is at best slight and variable, blocking of one such by an embolus or thrombosis renders the area of brain substance supplied by the vessel anæmic, and leads to complete or partial necrosis. The blocking by an embolus is followed by thrombosis in the vessel, so that, even if collateral circulation were possible, it would thus in a great measure be prevented.

The arteries which are most liable to be blocked by an embolus are the Sylvian or middle cerebral, because they are a direct continuation of the internal carotids. The left carotid artery, arising as it does direct from the arch of the aorta, is more likely to receive embolic particles than the right, and therefore embolic softening of the left hemisphere is more common than that of the right. Hilton Fagge throws doubt on this, believing that the explanation is—more cases of right hemiplegia from embolism have been published because of aphasia. The carotid itself is seldom blocked; the order of frequency of blocking of the cerebral vessels by embolism being—the middle cerebral, posterior cerebral, vertebral, anterior cerebral.

The results of embolism and thrombosis are essentially the same—the arterial blood supply is cut off, and there is anæmia of the area supplied by the artery. For the first twenty-four hours there is only a slight change in the appearance and consistence of the part, although I have found that the neurons may show microscopically well-marked histo-chemical changes. The area has generally a pale appearance; sometimes the capillaries may become distended by a backward flow of blood from the veins, and, giving way, produce small hæmorrhages into the perivascular lymphatics. Later on the tissue breaks down and softens, which is due to imbibition by the dead tissue of cerebro-spinal and serous fluids. The area of softening remains white, when very little blood returns from the veins to the capillaries; it is red, when blood does return from the veins, especially when the walls of the capillaries give way allowing the red corpuscles to escape.

Since the grey matter is far more vascular than the white, red softening is generally seen in the cortex and basal ganglia. Yellow softening is merely a later stage of the red, owing to alterations in the blood pigment.

Microscopically, the softened mass consists of myeline drops, swollen and degenerated nerve fibres, altered nerve cells, and granular corpuscles of Glüge with free fat granules. These granular corpuscles, which may measure as much as 30 μ, are leucocytes distended with fatty débris. If the circulation be re-established within a short time, the nervous structures do not necessarily die, but regeneration is impossible, if necrosis has commenced. Resorption of the dead tissue is gradually brought about, with eventual formation of a cyst. If the area is small, a scar of fibrous tissue may be the sole indication of the destruction that has taken place. The convolutions of the cortex, especially in senile atheroma, may sometimes be seen atrophied and sclerotic, and on tearing off the thickened pia arachnoid they present little erosions of a rusty yellow colour. Sometimes portions of the convolutions, or even a whole lobe, may have entirely disappeared, and the space be occupied by a serous fluid, covered superficially by the thickened pia arachnoid membrane.

Symptoms.—As a rule the onset in embolism is sudden, and without premonitory symptoms, and the patient, unless he be suffering with ulcerative endocarditis, is usually in good health; and if a large vessel, such as the middle cerebral, is blocked, consciousness is lost, and the symptoms may resemble an apoplectic attack, due to cerebral hæmorrhage; but although there may be coma, it is not usually so deep nor so lasting. Slowing of the pulse, a sign of increased intracranial pressure, is absent. Epileptiform convulsions are more generally met with, and, instead of the temperature falling, there is often in a day or two considerable pyrexia. The commonest valvular lesion connected with the production of embolism is mitral stenosis.

A gradual onset of symptoms in embolism may occur, owing to the impaction of a small embolus, which afterwards leads to considerable thrombosis. The most frequent result of embolism is hemiplegia, especially of the right side, associated with aphasia; and sometimes there may be word-blindness or word-deafness, and if the posterior cerebral be blocked, there is hemianopsy. Monoplegias are rare.

Thrombosis is usually preceded by premonitory symptoms, and often the patient is already known to be suffering with chronic brain disease, due to atheromatous or syphilitic arteries. In the majority of cases, headache, giddiness, formication, numbness, slight paresis of a limb, or one side of the body; loss of memory, irritability, amnesia, and slight speech defects, in persons past middle life, indicate degenerative changes of an advanced nature in the cerebral vessels. In syphilitic arteritis, the premonitory symptoms may be more acute and severe.

The onset is usually gradual, and for this reason is less often associated with coma, although sudden loss of consciousness, deepening to coma, by no means precludes the possibility of thrombosis. More frequently the hemiplegia precedes the coma, which is usually slight and less severe.

Vomiting is common in softening, especially in obstruction of the basilar artery.

One of the commonest results of extensive softening is dementia, which is usually accompanied by some paralysis. The patients have weakness of intellect, loss of memory, no idea of time or place, sometimes suffer with hallucinations and delirium, are generally emotional, laughing and crying without any reason for so doing, and always the subjects of obstinate constipation. Another result of softening, besides hemiplegia, is unilateral convulsions starting in the paralysed side; but, after a time, the fits may closely resemble idiopathic epilepsy.

Diagnosis.—In some cases it is impossible to tell whether a patient who has had an apoplectic seizure is suffering from embolism, thrombosis, or hæmorrhage. If a young person, suffering with valvular disease of the heart, is seized suddenly by loss of consciousness and hemiplegia, it is probably due to embolism. Evidence of embolism of other organs, such as the kidney and spleen, should be looked for. Sometimes there is blindness of one eye, owing to blocking of the arteria retinæ centralis. It must, however, be remembered that apoplexy due to hæmorrhage may occur in infective endocarditis, owing to rupture of an aneurysm, occasioned by previous embolic occlusion and infective inflammation of an artery.

If the patient is over 45, and the onset sudden, with deep and continuous loss of consciousness, and coma, flushed face, strong pulsating carotids, and slow pulse, it is more likely to be cerebral hæmorrhage; but it may be thrombosis of a large vessel, *e.g.* the basilar artery, in a person suffering with arterio-sclerosis, or syphilitic endarteritis.

The sudden onset and deep coma does not preclude thrombosis, although as a rule thrombotic softening is preceded by prodromal symptoms, and the onset is gradual. It must be remembered, moreover, that miliary aneurysms, the cause of cerebral hæmorrhage, are a part of a general, widespread arterial degeneration, so that the conditions relating to softening and hæmorrhage are frequently associated, except that in the former the general arterial pressure is usually not raised, whereas in the latter it is.

Epileptiform convulsions indicate softening rather than hæmorrhage; but the writer has seen two cases of hæmorrhage into the centrum ovale, in which there were epileptiform convulsions, with an incomplete loss of consciousness for some days before coma and death. One of the most important causes of thrombosis and softening is syphilitic arteritis, and a careful history should always be obtained from the patient or his friends, of symptoms pointing to specific infection. Even in the absence of any evidence, it is often advisable to diagnose syphilitic arteritis as the cause of an apoplectic attack in an adult male under 40. We must also bear in mind that rupture of a latent abscess, and the epileptiform and pseudo-apoplectiform seizures of general paralysis, may give rise to an apoplectic condition something like that due to vascular occlusion. For the diagnosis of the probable seat of the lesion, the reader is referred to p. 722.

Prognosis.—Recovery from the immediate danger depends upon the severity and the duration of the symptoms, on the occurrence of previous attacks, and the nature of the morbid process which has been the cause of the attack.

If the coma is deep, it indicates occlusion of a large vessel, and the prognosis is bad. The duration of the coma appears to be of less ominous significance than its intensity. A person may recover after four or five days' loss of consciousness, if the coma is not deep. Previous attacks increase the immediate danger, and also add to the probability of permanence of the symptoms, should the patient recover. The danger of vascular occlusion and softening is less from syphilitic endarteritis and embolism than in senile arterio-sclerosis, which is a progressive degenerative process, not benefited by treatment.

With regard to the paralysis and loss of function following softening from vascular occlusion, the prognosis will depend very much upon the improvement which is observable in the first week or two following the attack. A certain amount of repair may be looked upon as possible by the re-establishment of collateral circulation; but, when once softening has occurred, lasting for days, no treatment can restore the destroyed brain tissue, and all that can be hoped for, is compensation, by other structures taking on the function. Recurrence of the symptoms is more likely to occur in senile arterio-sclerosis; it may, and often does, occur in syphilitic arteritis, especially if it is not treated. Softening of the brain is a common cause of hemiplegia with contraction, post-hemiplegic athetoid spasm, aphasia, and other forms of speech defects, with or without dementia. Recurrence of the convulsions is not uncommon, and the writer has seen cases of softening in which the hemiplegia has persisted, but in which a form of epilepsy has developed; also cases in which patients had recovered from the paralysis, but subsequently suffered from fits like idiopathic epilepsy. The prognosis, as regards recovery or even improvement, in all such chronic conditions, is most unfavourable.

Treatment.—In the initial stage the treatment is the same as for apoplexy due to hæmorrhage, except that the purgatives administered should not be powerful. Diuretics may be administered with advantage, and if the heart be feeble and irregular, some stimulant and cardiac tonics may be given. Venesection should not be practised. It is better to avoid alcohol, as during the process of inflammatory softening it would tend to increase the inflammatory reaction. If the convulsions are severe, bromides may be given—if necessary, per rectum. If the thrombosis is of syphilitic origin, antisyphilitic remedies should be given. Iodide is of no good in embolism, and it is of doubtful use in senile thrombosis. The bowels should be attended to, the water drawn off, if necessary, although sometimes a hot sponge to the perineum will suffice, when there is retention. The patient should be placed upon a water-bed, and great care taken to prevent bedsores. A few weeks after the attack, gentle rubbing of the muscles of the limbs may be practised. It is not desirable to apply electricity till a month or six weeks after the attack. The only treatment for speech defects is educational.

F. W. MOTT.

HEMIPLEGIA.

By far the most frequent and most noticeable permanent symptom of cerebral hæmorrhage or softening is a unilateral paralysis, affecting one-half of the body; hemiplegia, due in the majority of cases to destructive or functional disturbance of the pyramidal system of neurons in the anterior two-thirds of the posterior limb of the internal capsule. The permanency of the paralysis depends upon two conditions—(1) the seat and the extent of destruction; (2) the amount of compensatory restoration of function by bilateral association and representation in the opposite hemisphere.

General Symptoms.—Before fully considering the special characters, and the symptoms and duration of cerebral hemiplegia, it will be well to discuss the two points alluded to—(1) A transient or temporary hemiplegia may result from lesions involving structures in the neighbourhood of the pyramidal system contained within the internal capsule, interfering by pressure or anæmia with the function of transmission of voluntary impulses from the motor cortex, but without destruction of the fibres themselves; (2) the hæmorrhage may be small and situated in (*a*) the anterior or (*b*) the posterior part of the hinder half of the capsule. In the former case, (*a*) the pyramidal system, which presides over the voluntary movements of the opposite side of the face, and to some extent of the opposite arm, would be permanently damaged, and there would be probably functional disturbance of the remainder of the arm and the leg fibres; in such a case, the leg would recover first, then the arm. Now, in the latter case, (*b*) where the lesion is situated in the posterior third of the hinder limb of the internal capsule, the sensory afferent system would be destroyed, and as a consequence there would be hemianæsthesia and sometimes a temporary hemianopsy. Reference to Fig. 18 shows that the leg fibres are the most posterior of the motor fibres. Consequently, when there is hemianæsthesia, they are more likely to be damaged, and, in such a case, the arm recovers movement before the leg.

Movements, and not individual muscles, are represented in the cortex cerebri. These voluntary movements are brought about by the correlative action of groups of muscles, one group often acting antagonistically to another group; *e.g.* in clenching the fist both the flexors of the fingers are in action, and also the extensors of the hand. The synergic action of muscles under the influence of the will, then, is represented in the cortex, and the paralysis due to cerebral lesions relates rather to the abolition of these synergic movements than to paralysis of muscles. For example, a hemiplegic man cannot move an arm by an effort of the will; but under a reflex spinal influence, such as yawning, both arms may be raised above the head in association. This offers an example of bilateral associated movement.

In hemiplegia, the arm, and particularly the hand, the movements of which are independent, and therefore more highly and specially represented in the cortex of the opposite hemisphere, is more paralysed than the leg or the face, which are to a great extent bilaterally associated. The upper muscles of the face are hardly at all affected, neither are the trunk muscles, because they act in bilateral association with the muscles of the opposite side.

Bilateral association can be explained by supposing that the centres which preside over such movements exist in both hemispheres: or a more probable explanation is, that the motor neurons which preside over the muscles which act in association are intimately connected by association fibres in the cerebral and spinal commissural tracts (Broadbent).

Special Symptoms.—The special characters of cerebral hemiplegia, whether arising from hæmorrhage or softening, may now be considered. When the patient has recovered from a state of unconsciousness or coma, there is generally found a paralysis of the opposite half of the body to the lesion. He complains usually, but slightly, of headache; there may be some defective memory and irritability of temper; there may be actual aphasia, or merely thickness of utterance. Convulsions are rare in hæmorrhage, frequently met with in softening, and optic neuritis is never seen in either.

Early Symptoms.—The appearance of the patient is generally such as to indicate paralysis of the face; there is, however, only paralysis in the lower half, indicated by the angle of the mouth being drawn over to the non-paralysed side; the forehead is wrinkled, and the eyes can be closed; but the patient is unable to wink the eye on the paralysed side, and sometimes he cannot close it independently of the other. He is unable to whistle, and food accumulates between the cheek and the lower jaw on the paralysed side; all voluntary actions of the mouth, *e.g.* showing the teeth, moving the mouth from side to side, exhibit the paralysis of the angle of the mouth more markedly than involuntary actions, such as laughing and yawning. Even in the lower part of the face, the loss of power is paresis rather than paralysis, but why the upper part of the face should escape has received no adequate explanation.

The tongue, when protruded, is projected towards the side of the paralysis, and away from the lesion. This is owing to the unopposed action of the genio-hyoglossus of the non-paralysed side, for both genio-hyoglossi acting together protrude the tongue in the middle line. The muscles of mastication are practically unaffected; there may be thickness of articulation and utterance in left hemiplegia (dysarthria), but this as a rule soon passes off; in right hemiplegia there is generally aphasia. There is often paresis of the soft palate, which hangs lower and moves less than on the sound side, and the uvula deviates slightly from the middle line; the most important paralytic phenomena, however, are exhibited by the arm and leg, although the respiratory muscles can often be found to be weaker on the paralysed side, when the patient wills to take a deep breath; possibly this may account for the fact that hypostatic pneumonia is more liable to occur on the paralysed side. Again, if the patient be made to sit up, the rectus abdominis is felt to be contracted less firmly on the paralysed side. The arm and leg are usually both paralysed, frequently to such a degree that no voluntary movement is possible in either limb; sometimes, however, it is only a hemiparesis, and occasionally the paralysis in the leg is more marked than in the arm, or *vice versâ*. The most frequent result of cerebral hæmorrhage is at first a complete paralysis in both arm and leg, and, in the course of time, a return of certain movements, but with permanent impairment of movement in the arm and hand of the paralysed side.

Initial rigidity may come on within a few hours of the onset, and probably is due to irritation of the lacerated pyramidal fibres. A few days after the apoplectic seizure, early rigidity appears in the paralysed limbs; it may be so slight as only to be apparent upon passive movement; it may be limited to muscles acting upon certain joints, or it may affect all the muscles of the arm and leg. Early rigidity denotes irritation, and is of unfavourable omen; it generally soon disappears; but it may persist for weeks, and pass into late rigidity. The muscles affected do not show any atrophic changes, and spasm is relaxed during sleep. Pain is caused if an attempt is made to overcome the rigidity by extension.

As a rule the superficial reflexes are diminished and the deep reflexes increased. The superficial skin reflexes, *e.g.* plantar, cremasteric, and abdominal, are usually diminished and sometimes abolished on the paralysed side; moreover, the conjunctival reflex is also considerably diminished on the paralysed side. In contra-distinction to the skin reflexes, the deep tendon reflexes are increased, so that the knee-jerk is exaggerated, and a wrist tap contraction may be obtained; not infrequently ankle-clonus can be produced ten days after the onset. Charcot taught that the increase of the tendon reflexes was due to degeneration of the pyramidal system in the spinal cord.

There may be a temporary blunting of sensation on the paralysed side; sometimes the apopleetic seizure may be preceded by paræsthesia in the limbs. Hemianæsthesia and a transient hemianopsy may occur when the posterior part of the internal capsule is damaged. It is rarely that permanent hemianopsy with hemiplegia occurs in cerebral hæmorrhage. The muscular sense may or may not be affected, but nothing very definite has been described.

Trophic and vasomotor changes in the paralysed part.—The skin of the paralysed side may be warmer by half a degree or more than on the healthy side. An acute bedsore may sometimes arise over the gluteal region of the paralysed side. It begins as an erythema, then a bleb forms which rapidly sloughs, and gangrene follows. It very seldom occurs if adequate precaution be taken; and the view of Charcot, that it is due to a purely trophic change, is not generally accepted.

The general nutrition of the patient is usually not affected. As soon as the patient recovers from the unconscious condition, there is power over the sphincters, but usually the bowels are constipated.

Later symptoms.—*Late rigidity and contracture.*—This is probably due to spinal and cerebellar over-action, owing to the loss of the normal cortico-cerebral control, by the destruction of the pyramidal system. A most characteristic feature is an increase of the deep reflexes and a certain attitude and gait. All associated movements return sooner than unilateral movements, consequently the leg recovers before the arm, and usually sufficiently to enable the patient to walk about with a stick; whereas the arm, especially the hand, may be permanently incapacitated. It is noteworthy that the apex of the limb which is most highly represented in the cerebral cortex is most affected, and is the last to recover movement, if it ever does. Secondary contracture or late rigidity is always most marked in the upper extremity, which is adducted at the shoulder, flexed at the elbow so as to resist all attempts at extension; the wrist is flexed upon the forearm, and the fingers upon the hand. It will be observed that in the upper extremity the flexors overcome the extensors, and the position of contracture is most characteristic. In the lower extremity the contracture is rarely so evident; the muscles in which the loss of power is most marked are the dorsal flexors of the foot and the flexors of the ankle; consequently, to prevent the toes from dragging on the ground, either the knee must be flexed so as to lift it from the ground, or, more commonly, the knee being extended by the quadriceps extensor, the difficulty is overcome by the patient swinging the foot round in the arc of a circle, the inner side of the toe of the boot scraping the ground. As contracture develops, there may be a considerable amount of pain. Disuse atrophy may occur, especially in hemiplegia of long standing; but the muscles retain their excitability to both faradic and galvanic currents. Occasionally a condition termed cerebral muscular atrophy may occur, the pathogenesis of which is not understood; but certain groups of muscles are truly atrophied, *e.g.* the interossei, the muscles of the ball of the thumb, etc. There may be swelling, sometimes painful, of one or more joints on the paralysed side.

Among the late secondary changes met with in hemiplegia, especially in the early or congenital form, are various disordered movements, *e.g.* tremor of the affected limbs, mobile spasm, athetosis, a term invented by Hammond to denote, without fixed position; post-hemiplegic chorea; and these various conditions are described in the chapter on Infantile Hemiplegia.

Psychical disturbances.—The patients are often emotional, and display various signs of mental weakness; they readily laugh or cry, and their memory may fail them, symptoms in some cases due, no doubt, to the associated disease of the cerebral vessels.

Treatment.—The friends should be warned that the chances of a complete recovery are remote; it may be safe in not a few cases to say that the patient may recover power in his leg sufficiently to enable him to walk; but there will be permanent impairment in the use of the hand, especially for all finer movements. Passive movements of the joints and massage of the limbs may be practised as soon as all irritative phenomena are over—that is, about three or four weeks after the onset. Electrical treatment by faradisation and labile galvanism may also be tried; likewise, systematic exercises by passive movements are often beneficial. Iodide of potassium and strychnine may be given internally, and the use of warm salt water baths is recommended. When there is late rigidity and contracture, little good can be done by electricity and medicines, and it will be the duty of the medical man to tell the patient and friends that there is no possible hope of cure, otherwise he will perchance fall into the hands of quacks.

F. W. MOTT.

INFANTILE HEMIPLEGIA AND DIPLEGIA.

Infantile paralysis of cerebral origin includes a large number of different types of motor disturbance. It would be more satisfactory if we could classify them according to their pathological cause, but in a large number we are as yet in the dark as to the etiology, and only a few post-mortems have been made in well observed cases.

Little in 1862 read a paper before the Obstetrical Society, in which he showed that a great number of these cases, especially of the diplegic form, were due to difficult labour. The cerebral diplegia of infancy has been termed Little's disease.

Strümpell in 1885 suggested that acute polio-encephalitis was the cause of a number of these cases. His views have not been universally accepted, but cases have been recorded which support them.

HEMIPLEGIA.

Etiology.—The statistics of 376 collected cases show that rather more than 5 per cent. were congenital. No etiological factor was discoverable in about half the cases. As to sex there is not much difference; some statistics show a slight preponderance of males, others of females. Previous health is not of much importance—strong as well as delicate children are affected about equally. In rather less than 10 per cent. there was a neuropathic history in the parents. Alcohol and syphilis in the parents are probably factors in some cases. All authors are agreed that it occurs most frequently in the first two years, and rarely after three years. The age at which it commences has no decided influence upon the severity of the disease.

Infectious diseases predispose to this condition, *e.g.* measles, scarlet fever, variola, rötheln, whooping cough, diphtheria, and typhoid. The disease may appear towards the end of scarlet fever. In 14 per cent. of Osler's cases there was a history of an infectious disease. Abercrombie describes four cases in which there was congenital syphilis, and Fournier looks upon the disease as a parasyphilitic affection. I have seen in the epileptic wards of the London asylums several cases of birth palsy, in which there were unmistakable signs of syphilis, *e.g.* chorio-retinitis. Possibly those cases in which no etiological factor can be found are really cases of polio-encephalitis of Strümpell. Injury of the head received at birth accounts for a large number of the cases. This occurs in lingering labours from unusual size of the head, from narrowing of the maternal passages, from breech presentations, etc., or may perhaps be directly due to the instrumental assistance for which those conditions call. But similar injury also occurs when the labour is unusually quick owing to forcible uterine contraction.

Morbid anatomy.—Cysts, areas of sclerosis or softening, may be found the result of embolism, thrombosis, or hæmorrhage.

Porencephalon is generally congenital rather than acquired It is a defect of the convolutions of variable extent, by which a cavity is formed penetrating a variable distance into the hemisphere, sometimes as far as the ventricles. The meninges are intact, and present neither thickenings nor adhesions; often the membranes are found adherent to the ependyma of the ventricle; the ventricular cavity communicates freely with the subarachnoid space, and the defect is generally limited to some definite vascular supply. Kundrat pointed out that the convolutions around present a radiate appearance.

Groups of convolutions, an entire lobe or the whole hemisphere, may be wasted, and usually in such cases there is atrophy of the opposite half of the cerebellum. The membranes may appear normal, more often they are thickened and adherent. Sometimes there are little nodular projections all over the surface of the atrophied convolutions. In a case of this disease, which I examined microscopically, the pyramidal tracts were entirely absent, and there were practically no pyramids in the medulla. In the spinal cord the pyramidal tracts were represented by little cleft-like indentations in the lateral column, so that not only did myelination not take place, but the pyramidal neurons must have been destroyed before birth. Microscopical examination of the cortex cerebri showed evidence of old and recent inflammation in the form of multiple hæmorrhages. The central convolutions were especially affected by the sclerosis. The most important morbid conditions are porencephalon, which may be congenital, or result from hæmorrhage at birth, and cortical sclerosis with atrophy. I think there can be no doubt that this condition is of inflammatory origin; but is it a primary inflammation, polio-encephalitis, as suggested by Strümpell, or is it secondary to thrombosis of veins, as Gowers suggests? Both theories still want proof—the multiple hæmorrhages, the atrophy of nervous tissue, and the overgrowth of glia tissue would be compatible with either.

Symptoms.—The onset is sudden in more than half the cases, according to Freud; there are convulsions, generally associated with loss of consciousness, which may last from a few hours to a few days. The convulsions may be general or unilateral. There is usually pyrexia, vomiting, and delirium. The paralysis may be observed after the first series of fits, or perhaps only a hemiparesis; but generally a fresh series of convulsions occur, and then there is complete hemiplegia. Frequently a considerable number of separate convulsive seizures occur before the paralysis sets in. Various authors differ in their statistics as regards the frequency with which the respective sides are affected; all, however, agree that the face is, as a rule, neither markedly nor permanently affected.

After weeks or months, in typical cases, an improvement sets in. A certain amount of movement returns, and the patient may learn to run about, and even recover some of the coarse movements of the arm. Muscular contracture and rigidity are especially liable to develop. The hemiplegia is like that in the adult, only the face and tongue are less affected. The arm, and especially the hand, is much more paralysed than the leg, and, owing to the bones and muscles not developing properly, in later life it is generally considerably smaller than its fellow. The facial paralysis is usually so slight that it is not perceptible at rest, and only when the patient laughs or cries does it become obvious. The irritation phenomena are more distinct in the face than the paralytic.

Late rigidity and contracture usually, but not invariably, occurs. The upper arm is adducted to the trunk, the forearm flexed, the hand extremely flexed, or extended; all the joints of the fingers may be flexed, and the hand closed in contracture, or they may be extended at the interphalangeal joints and even hyperextended so as to produce subluxation.

The tendon reflexes are increased, the knee-jerk is nearly always brisk or exaggerated, and ankle-clonus may be obtained. The paretic muscles are not necessarily uniformly affected by an increase of tonicity. Associated movements are more intense and more often met with than in the adult hemiplegia.

Post-hemiplegic disordered movements, probably irritation phenomena, are of special importance. These have been designated post-hemiplegic chorea, hemichorea, athetosis, hemiathetosis. These terms have given rise to a good deal of confusion, and it would perhaps be better to apply the term mobile spasm to all these forms, and divide them into two classes—(α) arising during attempt at voluntary movement, and consisting of choreiform, inco-ordinate movements—post-hemiplegic chorea, hemichorea; (β) involuntary and somewhat rhythmical movements occurring in the paralysed limbs, especially in the fingers and toes, generally persisting during sleep, and in rare instances affecting the muscles of the mouth. They are not of cortical origin, but due to lesions about the basal ganglia, *vide* Freud and Rie's case of athetosis, in which a calcified mass was found in the lenticular nucleus, and in other cases a tuberculous mass in this position has caused similar symptoms.

Spontaneous movements in orderly sequence may occur, the most common being slow opening and closing movements of the hand, the fingers being widely separated and hyperextended, also movements of supination and pronation, of abduction and adduction of the hand. These movements persist during rest and not infrequently during sleep. Slight differences can often be brought out, and frequently the movements are exaggerated by emotion.

The eye muscles are seldom affected. Sensibility is generally unaffected in infantile hemiplegia, even with athetosis. Sometimes the patients complain of pains on the paralysed side. Hemianopsy may occur, but is rare.

Aphasia was met with in 30 per cent. of left-sided cases, in 45 per cent. of right-sided. These figures, based upon an analysis of 300 published cases, indicate that just as there is a tendency in most people to specialise the use of the right hand by the left hemisphere, there is a tendency to specialise this half of the brain for speech; but the fact that the aphasia is not always permanent, shows that the other hemisphere can in early life take on the function.

Epilepsy is a very frequent and baneful result. In many cases the convulsions may be limited at first to the affected side; they are Jacksonian in type. Consciousness remains unaffected, the initial cry and biting of the tongue being absent; but they tend sooner or later to become generalised, and indistinguishable from true epilepsy. Attacks of *petit mal* are frequent, and, putting together the lowest, 25 per cent. (Osler), and highest, 75 per cent. (Gowers), proportional percentages, it may be roughly computed that one-half of the cases of infantile hemiplegia eventually develop epilepsy.

Mental defects may be, on the one hand, *primary*, and due to destruction or arrest of development of the brain, leading to all forms of mental defect, from feeble-mindedness to imbecility, from imbecility to idiocy; on the other hand, *secondary* to the epilepsy, and terminating in dementia. The child, in mild cases, usually learns to walk and to talk later, and generally displays backwardness in mental development. There are cases in which the rigidity and weakness tend to affect the opposite side; not infrequently athetosis comes on in the healthy side, although to a less degree. Those cases form a connecting link of transition to an important group of cases with bilateral symptoms.

DIPLEGIA.

A number of different pathological conditions, producing clinically bilateral symptoms, are included in this group. It may be the symptoms are those of—(1) general rigidity, (2) paraplegic rigidity, (3) spastic rigidity, (4) spastic diplegia (bilateral hemiplegia with rigidity), (5) general chorea, (6) bilateral athetosis. The character of the bilateral symptoms depend upon the nature and extent of the lesion. Owing to the injury being usually extensive and affecting both hemispheres, the mental condition is frequently profoundly affected, and the patients are generally helpless imbeciles or idiots. The affection is as a rule the result of congenital prenatal affections, *e.g.* porencephalon, or injury occurring during the short period between intra- and extra-uterine life. Breech presentations, forceps cases, and protracted labours can frequently be assigned as the cause.

Morbid anatomy and pathology.—Sometimes there is *agenesis corticalis*, failure of development of the convolutions and of the pyramidal system of fibres in the brain and cord, porencephaly, or fœtal meningo-encephalitis. The birth palsies are due to meningeal hæmorrhage (case of M'Nutt), resulting in chronic meningo-encephalitis, sclerosis, cysts, and partial atrophy. The bleeding in all probability usually comes from a ruptured vein during the asphyxial condition. Extra-uterine acquired causes may be exactly the same as those of infantile hemiplegia, only less frequently met with. It may follow infectious fevers, *e.g.* I have lately seen a case of spastic cerebral diplegia in a child of 5. The history was briefly that the child had measles, subsequently fits, but was not paralysed, as it could dance to the barrel organ in the street; a little later it took whooping cough, and was suddenly seized with convulsions, unconsciousness, and fever, from which it recovered, leaving a typical spastic paraplegia. Probably this was due to meningeal hæmorrhage during the fits of coughing.

Symptoms.—Nothing may be noticed, until the mother finds the child is unable to use its limbs properly. It does not begin to walk at the proper time, and on examination a stiffness of the limbs may be noticed. The rigidity as a rule is more marked and more persistent in the lower limbs, and there is frequently adduction spasm. When held up and the legs put on the ground, it will be noticed that the child rests on the tips of the toes and the inner surface of the feet, the legs being crossed or the knees touching one another. The nutrition of the muscles is usually unaffected. Sometimes the child can neither walk, stand, nor sit. Speech is often affected, the articulation difficult and slow. The arms, the speech, and the mental condition may, however, undergo considerable improvement; the legs, on the other hand, maintain their spastic condition for life. Associated with spastic paraplegic rigidity there may be general spasm and disorder of movement of the upper limbs, so that the case may be mistaken for hereditary ataxy, or disseminated sclerosis. Some of the cases looked upon as congenital chorea are probably of this nature; strabismus in these cases is frequently met with. In other cases, besides rigidity of the lower extremities there may be muscular wasting and trophic disturbance; sometimes there is bilateral spastic hemiplegia, muscular paralysis, with contraction and atrophy affecting both sides of the body, but perhaps unequally. Usually, owing to the extensive lesion, there is marked psychical derangement, but as a rule control over the sphincters is generally acquired earlier than in other cases of mental defect. Another very remarkable condition is bilateral athetosis, usually dating from infancy; cases have been described by Osler and Massolongo, the latter describes three cases in a family of five. The parents were healthy, and had no hereditary taint; the disease commenced in an identical manner in each case at the age of 7, with stuttering and loss of control over the tongue, spastic gait, and bilateral athetosis, affecting especially the hand; as each child grew older so the symptoms progressed. Oppenheim has described two cases, mother and child, of infantile pseudo-bulbar paralysis.

Family and hereditary forms of diplegia.—A number of cases of paralysis occurring in members of the same family and of different types have been described; some of these are more fully described in the section on the Spinal Cord, *e.g.* hereditary ataxy and ataxic paraplegia.

The symptoms of these diseases depend in a great measure upon the systems of neurons that are affected. We shall now briefly refer to a few of them. A severe form of infantile paralysis of cerebral origin associated with blindness, first observed by Waren Tay, and termed by Sachs, who first described its pathology, amaurotic family idiocy. Several children of the same family or having blood relationship, with no history of hereditary syphilis and otherwise normal, are affected by a remarkable and characteristic group of symptoms, which run a rapid course and terminate in death by marasmus at the end of about two years. These cases have been found among Jews. There is absence of mental development, and idiocy, associated with a progressive amaurosis, which terminates in complete blindness and white atrophy of the optic nerves, more or less spastic paralysis of the extremities, with frequently nystagmus and strabismus. Both Sachs, and Kingdon and Russell have described the macroscopical and microscopical changes in the nervous system. As there was no evidence of vascular change, or of inflammatory processes, we must therefore assume that the disease is an hereditary failure of development, *agenesis corticalis*, as it is termed by Sachs; Russell, however, thinks that the recent degenerative changes are of toxic origin. Spastic paraplegia is usually described in the section of the spinal cord, but it may be a disease affecting primarily the cerebral pyramidal neurons. Frequently members of the same family may be affected by spastic paralysis, often associated with nystagmus, slow scanning speech, and mental defects, thus resembling hereditary ataxy, except that the knee-jerks are present or increased, and there is no oscillation in the gait.

In a disease described by Charcot and Marie, very similar to Friedreich's disease, there are all these symptoms present; but, besides, there are various affections of the ocular muscles and visual defects, *e.g.* ptosis, paralysis of the external rectus, absence of pupil reflexes, colour blindness, limitation of the field of vision, and white atrophy of the discs. In fact, these groups are closely akin; but in cerebello heredo ataxy there is no scoliosis and no club-foot, and optic atrophy is common. In hereditary spastic paraplegia there is degeneration of the cerebral neurons. In Charcot-Marie disease there is associated atrophy of the cerebellum.

Prognosis and treatment.—As infantile cerebral paralysis includes an "olla podrida" of diseases, and since most of them come under our notice when the mischief has been done, treatment of a curative nature can seldom be applied; yet in a well-marked hemiplegia, coming on suddenly in an infant, with the possibility of meningeal hæmorrhage and formation of a blood cyst on the brain, the symptoms might well point to early surgical interference. Cases of successful trephining have been reported; but as a rule the operation is resorted to when too late, and the brain substance has been destroyed or development prevented. Moreover, epilepsy has usually become permanently installed in most of these cases, and the operation, when performed with the object of preventing or diminishing the fits, fails.

If called to a case of convulsions in an infant, more severe and protracted than usual, with fever and other symptoms of severe constitutional disturbance, it is advisable not to forget the possibility of oncoming paralysis. Bromides, or even inhalation of chloroform, should be administered, if the former fail to check the convulsions (for it must be remembered that a venous hæmorrhage is more likely to occur in the asphyxial condition); also a purge should be given. Birth palsies are usually not discovered for some time, but difficult and prolonged labour is just as likely to give rise to injury by venous congestion and rupture, as the use of the forceps; and no doubt practitioners have been unfairly blamed for using these instruments, when really the cause was the condition which necessitated their application. Still, unskilful and rough use of instruments does cause brain injury. When the paralysis is established, not much can be done by medicines. The *vis medicatrix naturæ*, aided by massage and passive movements, good feeding, and careful hygienic precautions, such as open air exercise, warm salt water baths, and, if necessary, surgical measures or mechanical contrivances of various kinds, to correct the deformities and contracture, may be applied. As a rule, the leg and face recover in great measure, and the aphasia is usually not permanent; but the hand and arm are nearly always permanently affected with contracture and paralysis, and there is generally wasting and mal-development, not unfrequently associated with mobile spasm. Of even more importance is the tendency to feeble-mindedness, idiocy, and imbecility, associated generally with unilateral epileptiform convulsions or true epilepsy, which is obstinate and usually does not admit of cure, although remissions and periods of quiescence do occur under appropriate treatment. Bromides, as a rule, are useless for the Jacksonian epileptiform convulsions. These patients are particularly liable to constipation, and if this be not prevented by laxatives and purgatives, fits are more liable to occur. Probably the best thing to do for the mental condition is to place the child in a special institution for the training of backward and feeble-minded children.

F. W. MOTT.

CEREBRAL TUMOURS.

Syn., Ger., *Gehirngeschwülste;* Fr., *Tumeurs cérébrales.*

Etiology.—The cause of cerebral tumours is as a rule unknown, except that of the syphilitic, tuberculous, and parasitic cyst varieties.

There are, however, a number of tumours of which glioma is the commonest, which commence in epiblastic structures; and these, together with other primary forms, are due to developmental causes as yet little understood. Cerebral tumour is not an infrequent cause of death; for, according to Starr, about one case in 120 is met with upon the post-mortem table of hospitals. Tumour is met with most frequently in early and middle life; it is twice as frequent in males as in females, and of all the cases of cerebral tumour more than one-half are tuberculous, Excepting syphilis and tubercle, the other forms of primary tumour may develop in apparently healthy persons, although frequently there is a history of a blow on the head or local injury. It may be that the blow merely excites inflammatory change in the tissue around a growth which is more or less latent, *e.g.* tuberculous deposit.

Morbid anatomy.—**Gliomata.**—The starting point of these tumours is neuroglia, the supporting structure of the central nervous system; they develop more frequently in the brain than in the spinal cord, and consist of cells and fibres which cannot be distinguished from the normal glia tissue, and therefore the morbid growth appears to be an active formative proliferation of this tissue; it is usually infiltrating, has no defined border, and causes increase of size in the portion of brain affected. Transverse sections of gliomatous brain appear grey or greyish red; the substance is soft and usually very vascular; the dilated vessels frequently give way, causing hæmorrhage and producing important clinical symptoms. The growth does not necessarily destroy the brain substance, for frequently the nerve fibres, being merely pushed aside, retain their conducting power. This form of growth is most frequently met with in the cortex cerebri and in the white substance of the centrum ovale, but also in the cerebellum, central ganglia, pons, or medulla. In nine cases out of ten the gliomatous tumour is solitary, and it is rarely adherent to the dura mater.

Sarcomata seldom arise in the brain substance, since they are of mesoblastic origin. Primary sarcomata commence in the fibrous structures of surrounding tissues, for example, in the membranes of the brain, in the dura mater, in the periosteum of the cranial bones, and in the bones themselves, especially of the base of the skull. They may be hard or soft, generally nodular, and they may consist of round cells or spindle cells; sometimes they are slow growing and fibrous, and frequently contain cysts from myxomatous degeneration. Rarely chondro-sarcomata or myxochondrosarcomata are met with. Metastatic deposits of sarcoma may occur in the brain.

Syphilomata.—These are described in the section on "Brain Syphilis."

Tubercle.—This form of tumour may be solitary, but, as a rule, like the tumours due to syphilis, it is multiple, and may vary in size from a pea to a filbert or chestnut. Tuberculous tumours are most frequently met with, according to Gowers, in (1) the cerebellum, (2) the cortex, (3) the pons. On transverse section, they have a sharply-defined border, and a yellow caseous appearance; they consist of granulation tissue, undergoing necrotic changes in the centre. To the naked eye they closely resemble gummata; but microscopically they are distinguishable by the presence of tubercle bacilli, which are frequently found in giant cells. They occur especially in children, and frequently give rise to hydrocephalus.

Carcinoma is nearly always secondary, and in the cases I have seen the primary growth has been usually in the mammary gland; this form is multiple, and grows rapidly.

Rare forms of tumour.—Psammomata are slow growing, and as a rule do not produce the general symptoms of intracranial pressure. I have seen numerous cases of slow growing tumours upon the post-mortem table in patients who were classed as epileptic dements in the asylums. These tumours are endotheliomata or fibromata; they contain a number of bodies like bird's-nest cells, which consist of calcareous concretions. They grate on section, and are usually very tough, not vascular; some of them appear to spring from the Pacchionian bodies, and I have seen them as large as a Tangerine orange. They are circumscribed, and indent, but do not infiltrate the subjacent brain tissue, and are therefore quite capable of removal. Cholesteatomata, angio-sarcomata, angiomata, dermoid cysts, cysts of the choroid plexus, and other growths, *e.g.* tumours of the hypophysis cerebri, are in rare cases met with.

Parasitic cysts (*Echinococci and cysticerci*).—The former are common in Australia, and occasionally occur in England; the latter are uncommon in this country, but are frequently met with in Germany and on the continent.

The pathological effects of tumours are direct irritation or destruction of nervous elements; increased intracranial pressure.

Symptoms.—The general clinical phenomena of tumours are due to increased intracranial pressure and irritation. As a rule the general symptoms precede the local, and the onset of both is slow and gradual: sometimes the symptoms have been so ill-defined as to have been overlooked during life, and the condition has only been discovered post-mortem.

General symptoms.—*Headache* is one of the earliest, most constant, and most distressing of symptoms, and is due to increased intracranial pressure; it may be dull, aching continuously, affecting the whole head, and exaggerated by every forced act of expiration such as coughing or sneezing. This general pain may be associated with very acute, excruciating, burning local pain, due to meningeal irritation. Frequently the principal seat of the pain is the region of the tumour, but by no means is this always the case. There is often local tenderness on deep pressure or percussion. In the case of syphilis, the pain is worse at night, and not infrequently awakens the patient at a particular hour. Macewen has pointed out that the note obtained by percussion over the seat of the tumour is high-pitched and somewhat resonant. Headache usually persists throughout the disease, and even in the stuporose condition the patient groans and puts his hands to his head, from which it may be inferred that there is still agonising pain.

Ophthalmoscopic changes.—One of the most important clinical symptoms of cerebral tumours is optic neuritis. It occurs before the death of the patient in at least four-fifths of the cases, is generally bilateral, but usually one eye is affected more than the other. It may be present without the patient complaining of affection of sight; though sometimes there is amblyopia. It is less frequently present when the tumour is situated in the membranes than when the brain tissue is involved. The intensity of optic neuritis depends upon the seat and rapidity of the growth of the tumour, rather than upon its size.

The cause of changes in the fundus, which English authorities speak of as optic neuritis and German as Staunng's papille, or choked disc, is a matter of dispute. There are three views—(1) That it is due to irritation and inflammation of the sheath of the optic nerve, produced either by irritation arising from substances contained in the cerebrospinal fluid of the sheath of the optic nerve, or by direct extension of meningitis. (2) That it is due to obstruction of the outflow of venous blood from the ophthalmic vein, by the increased intracranial pressure on the cavernous sinus; but this theory of von Graefe has been disproved by the fact that a free anastomosis occurs between the ophthalmic and facial veins. (3) That the intracranial pressure interferes with the return of lymph from the sheath of the optic nerve, causing œdema and swelling of the disc. Gowers seems to think that a combination of causes may be in operation. No doubt stasis in the veins would soon be followed by exudation and leucocytic migration, and then all the appearances of inflammation would be produced. In many cases of tumour, an ampullary swelling has been observed where the sheath is weak at the entrance to the eyeball; but it is often absent even in acute cases.

Vomiting is a frequent but not constant symptom; it is, however, one of the earliest and most persistent, and is frequently associated with giddiness; it may come on when there is no food in the stomach, especially when the headache has been severe. If the vertigo is very severe, it points to cerebellar disease; in fact, vomiting and vertigo are both more liable to occur in a severe form, when the tumour is situated about the base of the brain.

Infrequent pulse, 50 to 60, is often met with, and is due probably to increased intracranial pressure.

Fainting fits and apoplectic seizures produced by hæmorrhage may occur, causing sudden increase of intracranial pressure.

Epileptiform convulsions.—This symptom is often absent. When present, it indicates affection of the central convolutions; the convulsions are Jacksonian in type, unilateral, and frequently followed by loss of consciousness, usually preceded by a tingling and numbness of the skin of the parts in which the muscular tonic and clonic spasms commence. The convulsions spread in a definite march, but after a long time, in slow growing tumours (as Oppenheim has pointed out, and this I can verify from experience), symptoms like idiopathic epilepsy may occur.

Psychical symptoms.—The expression of the patient is often characteristic of a profound mental change. There is a dull, heavy, drowsy, or stuporose expression, the speech is slow, and sometimes slurred and confluent; the reaction time in answering questions is much prolonged; there is loss of memory of recent events, and sometimes delusions and hallucinations, and even no idea of time or place. Sometimes tumours are associated with symptoms which may be mistaken for hysteria.

General wasting and bodily weakness occurs, partly due to the vomiting, the pain, want of sleep, and the knowledge too often of a fatal and incurable disease; producing a marked depression of the metabolism of the whole body, which in a measure is increased by auto-intoxication, brought about by obstinate constipation.

General convulsions, resembling true epilepsy, or attacks of brief loss of consciousness, resembling *petit mal*, may occur in tumours of various situations having no special localising significance; and in the early stage of cortical tumours, general convulsions may occur which are afterwards replaced by unilateral fits.

Regional or focal symptoms.—Focal symptoms are caused either by direct or indirect involvement of structures with particular functions. The tumour may produce irritative phenomena, *e.g.* convulsions, or it may cause loss or impairment of function by destruction of the nervous elements.

Tumours of the hemispheres.—The central convolutions of the Rolandic region of the cortex may be the seat of the tumour, and produce two classes of localising symptoms—(*a*) irritative, (*b*) destructive. The former are characterised by unilateral convulsions, frequently commencing by a numbness and tingling in the skin, and accompanying or following the tonic and clonic spasm in the muscles of the part; the exact determination of the seat of the numbness and tingling, and where the muscular spasm commences, is an important indication for localising the seat of the tumour. The march of the fit is also of great importance to observe, for it will always commence in the same region and spread in the same way: *e.g.* it may begin in the face, indicating involvement of the lower portions of the Rolandic region; in the fingers, wrist, thumb, or shoulder, indicating the middle portion; or in the toes, ankle, or leg, indicating the upper portion of the central convolutions. The clonic spasms usually spread over the whole half of the body, and may extend to the other side and become general. Consciousness may remain throughout the fit or at least at the commencement. It is usually best if the fit spreads to the other side. It is important to determine the state of the part after the convulsion, and to note whether there is paresis or anæsthesia. Destructive lesions produce hemiplegia; but usually this commences with monoplegia of face, arm, or leg, and then paresis of the other parts, which eventually may cause a complete hemiplegia, as the nervous structures presiding over voluntary movements are successively destroyed. Partial or complete aphasia may occur if Broca's convolution or the auditory word centre be involved.

Prefrontal region.—Bruns has pointed out that tumours of the frontal lobe often cause ataxy of a cerebellar type, and thus tend to simulate cerebellar tumours; but frontal ataxia is often associated with hemiparesis, monoparesis, or partial aphasia; optic neuritis occurs later in the course of the disease, and mental dulness, stupor, and even appearances resembling imbecility, are early symptoms.

Parieto-occipital region.—Tumours may attain a considerable size in this region, without producing definite localising symptoms. Sometimes word-blindness and mind-blindness may occur when the left hemisphere is involved. If the occipital lobe be destroyed, hemianopsy would be the result.

Temporal lobe.—Tumours of the temporal lobe often cause no definite localising symptoms, but if on the left side it is possible for word-deafness to arise.

Basal ganglia.—Tumours in the corpus striatum may occur without producing any definite symptoms; but hemiplegic symptoms may occur from direct or indirect involvement of the internal capsule. Tumours of the optic thalamus may, however, be associated with hemianæsthesia and hemianopsy, from involvement of the optic radiations. Tumours in the neighbourhood of the third ventricle may present no definite localising symptoms, but the general symptoms may be very manifest, such as amaurosis, headache, dulness, hebetude, and vomiting (often pronounced); it is said that these symptoms are due to increase of intracranial pressure from compression of the veins of Galen and distension of the ventricles of the brain. Three cases of tumour of the third ventricle have recently come under my observation. They presented symptoms which could best be explained by supposing that accumulation of fluid in the ventricles was the cause. The main symptoms were recurrent attacks of drowsy stupor which sometimes deepened to coma, preceded by restlessness and sometimes by convulsions, general paresis, dementia, and optic neuritis.

Cerebellum.—The symptoms of tumours in this situation are ataxy, characterised by a peculiar oscillating gait, nystagmus, vertigo, and tendency to fall in one direction, muscular weakness on the same side as the lobe of the cerebellum affected, headache (especially over the back of the head), combined with rigidity of the neck. Vomiting and slow pulse usually occur; and whereas optic neuritis develops early and is very marked, mental symptoms in the form of apathy, dulness, and stupor develop late according to Bruns, affording thus a contra-distinction to frontal tumours.

Tumours at the base of the brain are frequently met with, and they are usually syphilitic, tuberculous, or sarcomatous, and other growths springing from the periosteum or bones of the skull. The cranial nerves are especially liable to be affected, and particularly the nerves to the eye muscles, producing strabismus, ptosis, or diplopia. The optic tract may be compressed and cause hemianopsy, or there may be pressure on one optic nerve, when there will exist unilateral symptoms and changes in the fundus; or the chiasma may be pressed upon, as in tumours of the hypophysis cerebri, and there will be bitemporal hemianopsy. The fifth nerve may be involved, causing anæsthesia of the face, loss of taste and smell on one side, and perhaps sloughing of the cornea. The facial nerve may be involved and give rise to peripheral facial paralysis, and the muscles of the upper part of the face will be involved as well as the lower, the eye will remain open in sleep, and the muscles will show quantitative and qualitative changes in the electrical reactions. Frequently, from pressure upon the crus, pons, or medulla, there may be weakness or paralysis of the extremities.

Crus cerebri or corpora quadrigemina.—The origin of the third nerve is very liable to be involved by tumours, and if there is the syndroma of Weber, namely, paralysis of the motor oculi of one side and complete hemiplegia of face, tongue, and limbs of the opposite side, the diagnosis is easy. If the corpora quadrigemina are involved, there is nystagmus, ataxy, and generally loss of pupil reaction.

Pons and medulla.—The symptoms of a tumour here depend upon involvement of the afferent and efferent projection systems, with generally one or more of the cranial nerves, *e.g.* the facial or the fifth of one side is paralysed, associated with loss of power in the limbs; if of the opposite side, it is termed alternate paralysis. For further particulars concerning regional diagnosis in organic diseases of the brain, see the special chapter, p. 655.

Diagnosis.—Three points must be considered—(1) Is the case one of cerebral tumour? (2) Where is it situated? (3) What is its probable nature? The first question can probably be answered in the affirmative when there is headache, vomiting, optic neuritis, and slow pulse. The second necessitates a consideration of the localising symptoms already discussed; and the third may be sometimes correctly diagnosed or guessed at by considering the history of the onset, progress, previous health, sex, and age of the patient. The diseases for which cerebral tumours might be mistaken are—(1) cerebral abscess, (2) hysteria, (3) general paralysis, (4) meningitis, (5) uræmia, (6) disseminated sclerosis. The optic neuritis of cerebral tumour is generally much more pronounced than in cerebral abscess or meningitis; moreover, other symptoms would help the diagnosis, such as the existence of ear disease and pyrexia. Certain cases of chronic basic meningitis may closely resemble cerebral tumour, likewise chronic hydrocephalus; and the cerebral symptoms which sometimes occur in lead poisoning, namely, headache, optic neuritis, delirium and convulsions, might lead to a mistaken diagnosis.

Prognosis.—If we can exclude the possibility of syphilis, the prognosis is most unfavourable. As a general rule, death takes place in one or two years. Occasionally tuberculous tumours become stationary; but in other tumours, when once the symptoms have become pronounced, death usually takes place from exhaustion, coma, or intercurrent disease in a comparatively short time, but death may sometimes be quite sudden and unexpected. Fibromata and endotheliomata, with calcareous concretions springing from the dura mater, are apparently much more chronic in their course. I have seen cases which have lasted for very many years.

Treatment.—**General management.**—It is well in all cases to try mercurial inunction and large doses of iodide of potassium, upon the chance that the tumour may be syphilitic, even if the patient denies that he has had the disease. In some cases I have found mercurial inunction over the painful spot in the head has given relief. Allen Starr has found that large doses of iodide of potassium give relief in non-syphilitic growths: possibly it relieves intracranial pressure. Liq. arsenicalis may be tried, and if the tumour is, as in all probability, tuberculous, cod-liver oil and tonics: for the relief of the pain, morphine often must be given hypodermically; and opium smoking might be tried. Gowers recommends cannabis indica as a narcotic. The pain sometimes is relieved by antipyrine and phenacetin, but as a rule these are not so efficient as morphine. Leeches or ice-bags to the painful part, accompanied by hot bottles to the feet and purgatives, may tend to relieve intracranial pressure, by drawing the blood from the head. The convulsions as a rule are not much relieved by bromides; chloral appears to be more effective, and chloroform inhalations in some cases have to be resorted to. The vomiting may be most troublesome, and may necessitate feeding by the bowel; ice can be sucked, opium given, and rest in bed upon the back should be enjoined.

Operative measures.—In 1885, Hughes Bennett and Godlee first removed a cerebral tumour; since then the operation has been performed successfully by Macewen, Horsley, and many others. The results, however, are not so satisfactory as at first this branch of surgery promised. Many patients die of shock; in few cases only can the tumour be removed without risk of return; often the patient is left paralysed or speechless or mentally afflicted; if the wound becomes septic, hernia cerebri is very likely to occur, also meningitis. In making up one's mind to recommend operation, we should be guided by the following considerations:—(1) That, according to Allen Starr, only 7 per cent. of cerebral tumours are suitable for operation. (2) Are we sure of correct localisation? When the tumour is in the Rolandic region, it can usually be localised by the character of the convulsions or paralysis, and if the classical symptoms of tumour are present it is better to operate at once. In some cases, even if there is not much chance of removal of the tumour, surgical interference may relieve intracranial pressure, and many of the distressing general symptoms arising therefrom; the optic neuritis may rapidly subside and post-neuritic atrophy and blindness thus be prevented. Allen Starr, who has made an exhaustive study of this subject, very properly says "that it is essential to success that these operations should be done in a hospital, and should be done only by those who have had considerable practice in the performance of these operations, both upon the cadaver and the living subject. In this way delay is avoided; shock, which is the most dreaded feature, is obviated; and the many complicating accidents of hæmorrhage, etc., are more skilfully met."

F. W. MOTT.

BRAIN SYPHILIS.

Syn., Ger., *Gehirn-Syphilis;* Fr., *Syphilis cérébrale.*

Syphilis is one of the most important factors in the production of organic disease of the brain. The virus appears to act in two ways—(1) directly upon the blood vessels, membranes, and connective tissues generally, with secondary destructive changes in the nervous tissue—a true specific inflammation; (2) by a direct influence upon the vitality and durability of the neurons themselves, producing systemic degenerative changes, of which general paralysis and tabes dorsalis are by far the most common and important. These are spoken of frequently as parasyphilitic (Fournier), and are treated of elsewhere.

Brain disease is among the common sequelæ of syphilis, and, according to Hjellman, about 2 per cent. of all the cases of syphilis, excluding parasyphilitic disease, are followed by brain lesions. A frequent result is an inflammation of the arteries, especially about the base, causing softening by occlusion through the specific inflammation, or more often by the secondary thrombosis. This arterial affection was recognised by Morgagni, but did not attract general attention until the classical paper written by Heubner in 1874 appeared; although numerous physicians had already described the morbid anatomy, and Clifford Allbutt had elaborated the microscopical anatomy.

Another very common result of syphilis is a local or general inflammation of the membranes, gummatous meningitis, and the formation of neoplastic deposits (gummata) on the surface or in the substance of the brain. Each of these cerebral forms of the disease may produce most varied symptoms: partial or complete occlusion of the vessels may cut off the blood supply from various portions of the brain, causing softening and loss of function (if there is complete occlusion), and disturbance of function, temporary or permanent, according as to whether there is compensatory supply of blood to the part by other vessels or not. With this condition alone it can readily be understood how protean in its aspect this disease may become, when we consider the varied special functions of different portions of the brain. This disease of the arteries may exist alone without any symptoms of cerebral irritation or increased intracranial pressure—conditions which are met with respectively in the two next varieties.

When the membranes are affected by the inflammatory process, the vessels are generally also affected; but not necessarily, so that we may have a combination of the effects of cerebral softening and vascular occlusion with cortical irritation or cranial nerve paralysis from gummatous meningitis. The irritation phenomena are pain in the head, specially worse at night, sometimes vomiting and convulsions, and other irritative phenomena, according to the situation of the lesion. Owing to the predilection with which the base of the brain is affected, paralysis of the cranial nerves is one of the most common results. The meningitis, in rare cases, may extend to the whole cerebro-spinal axis.

Lastly, there is besides irritative phenomena, increased intracranial pressure, owing to the formation of syphilitic deposits in the membranes extending into the brain substance, producing gummatous tumour or tumours.

Etiology.—Organic brain disease from syphilis may appear at any time from three months after infection to as long as twenty-five or even more years. Formerly it was believed that the disease was essentially a tertiary lesion; but one now knows that it frequently occurs within the first year after infection. In forty cases which have come under my observation, of which a history of syphilis and the time which has elapsed between the onset of the cerebral symptoms and the existence of the primary sore has been determined, it was found that four occurred in the first year, seven in the second year, six in the third year, and three in the fourth year, and the remainder at subsequent periods. One case occurred within six months, and some of the severest cases were those which occurred within the first few years.

Sex and age.—The disease affects particularly males between 20 and 45 years of age, because syphilis is more common in men, and because during this period of life infection is more likely to take place.

Heredity.—Families in whom there has been no trace of syphilis are probably more prone to severe sequelæ, owing to lack of immunity; and hereditary predisposition seems to play a part. I have had under my care two brothers, who, at nearly the same period after infection, developed almost identical symptoms of a cerebral gumma; both were treated with antisyphilitic remedies, and made good recoveries.

He has also seen cases of severe cerebral syphilis follow blows on the head, exposure to the sun, mental excitement, excesses *in Baccho et venere*, chronic lead poisoning, each of which in the respective cases appeared to be an exciting or predisposing factor. Of all the factors, however, which predispose to brain disease, lack of treatment or insufficiency of the same appears to be the most important. In my experience at the London County Asylums, where a large number of patients are admitted suffering with cerebral symptoms, few seem to have been adequately treated. I have, however, met with several cases which have been treated from the very first, and yet severe cerebral syphilis has occurred. Barlow pointed out that the subjects of congenital syphilis are also liable to brain disease, and present similar, although not absolutely the same, clinical phenomena as in the acquired form. Seeing that syphilis is more common in cities and large towns, this form of brain disease is much more frequently met with in urban than rural populations. .

Morbid anatomy.—The virus of syphilis affects the mesoblastic structures of the brain, and produces a round-celled infiltration of the membranes, which may form a superficial, colloidal, or gelatinous layer, or a deposit in the form of a node or nodule, or multiple nodes or nodules, and this round-celled infiltration may extend from the surface into the substance of the brain along the course of the vessels, or into the fissures, filling them up with a gummy mass. It is probable, as Wilks pointed out, that gummata do not begin primarily in the substance of the brain, but are extensions of the neoplastic formation from the surface along the vessels.

Naked-eye appearances. — The membranes thus affected appear thickened locally or generally, the condition depending upon the age of the process. If it is of recent origin, the inflammatory deposit may be soft and gelatinous, or greyish red, or of a yellowish colour; if of some standing, the inflammatory process may have gone on to the formation of scar tissue. Should the granulation tissue form a tumour on the surface or in the substance of the brain, a gumma as distinguished from superficial gummatous meningitis occurs; which, on cutting into, frequently presents in the centre or in some part of it a yellow appearance due to caseation, the surrounding parts being grey or greyish red, soft and vascular. Gummatous tumours may vary in size from a cherry-stone to a pigeon's egg.

Gummata are particularly common about the base of the brain in the neighbourhood of the optic chiasma, but they may occur in any part, and the writer has seen them involve the dura mater and erode the skull, projecting through externally. The gumma belongs to the granulation tumours (Virchow), and one may find in a single tumour all stages of the process, from the formation of the new tissue to caseation and cicatricial connective tissue.

The arteries present little greyish white nodules on one side usually, so that, when cut across transversely, the nodules present a half-moon appearance. The vessels themselves feel stiff and cartilaginous between the fingers, and will not collapse on pressure. When there is universal arteritis, the vessels, small as well as large, appear opaque, dirty white in colour, and their walls thickened, so that they can be cut easily transversely, owing to the resistance they offer. The vessels about the base are particularly liable to this inflammatory change; possibly it is due to the cerebro-spinal fluid, which exists there in abundance, and which possibly contains the toxine.

Microscopical appearances.—If gummatous meningitis be examined microscopically, the following conditions will be seen:—A round-celled infiltration of the tissues, increased vascularity, and changes in the vessel walls. These are infiltrated with leucocytes, and greatly thickened; the arteries generally show also a proliferation of the subendothelial layer of the intima. In a section of the gumma we find the same round-celled infiltration, with increased vascularity and changes in the vessels; but besides this, in the older parts of the growth, necrobiotic changes are seen in the cells, which may lead to their destruction; there is thus an amorphous mass which does not stain with logwood, and which may even have gone on to caseation. The cause of this necrotic change is obviously the simultaneous obliteration of the vessels by the inflammatory process. Sometimes cicatricial tissue is found.

Syphilitic arteritis is characterised microscopically by a cellular proliferation of the subendothelial layer. It generally affects one side of the vessel, but it may affect the whole of the intima. This endarteritis is frequently associated with a periarteritis. The inner coat is thickened, owing to a development of spindle-shaped and stellate cells. According to Heubner, these do not undergo caseous degeneration, and he distinguishes it thus from atheroma. There is actually nothing specific in the process, but it is strong presumptive evidence of syphilis when one finds a thickening of the endarterium, which has not undergone a caseating process. When the arteritis is very acute, a new formation of capillaries in the intima may take place.

Thrombosis of the diseased vessel is frequent; subsequent organisation of the clot may occur, and eventually the diseased vessel may be converted into a fibrous cord.

Vascular rupture is rare, so also is the formation of aneurysm. The important clinical result of endarteritis syphilitica is cerebral softening. The arteriæ fossæ Sylvii are most frequently diseased, so one finds syphilitic brain softening more often in the area supplied by these vessels than elsewhere.

Symptoms.—Allusion has been made to the general symptomatology in the introduction; we shall now consider certain special types of brain syphilis.

Basal meningitis, including new growths.—The clinical symptoms are pains in the head, usually worse at night, stiffness of the neck, disturbance of consciousness, giddiness, vomiting, fainting fits, and convulsive seizures. Mental symptoms often occur, *e.g.* confusion of ideas, enfeebled intelligence, and loss of memory. Sometimes the patient falls into a stuporose condition; he may, on the one hand, lose consciousness, and eventually become comatose; on the other hand, maniacal symptoms, with delusions, may arise. Not infrequently there is polydipsia and polyuria. There is great variability not only in the opinion of authors as to the existence of pyrexia or not, but in the character of the fever itself, when it does occur. It is, however, pretty generally recognised that syphilitic meningitis differs from other forms of meningitis, *e.g.* tuberculous by the absence of pyrexia. If pyrexia be present, it is usually of an anomalous form; it may be remittent, and sometimes at the onset of the disease there may be a rise of temperature in the evening; but in my experience, pyrexia is quite unusual in syphilitic brain disease.

Disturbances of vision.—Limitation of the field, hemianopsy, and unilateral or complete blindness, may result from affection of the chiasma or optic nerves, through direct involvement by the disease, or by increased intracranial pressure. Ophthalmoscopic examination often reveals optic neuritis or optic atrophy. Probably the most frequent result of all is a paralysis of eye muscles, causing ptosis, squint, and diplopia. The third nerve is especially liable to be affected, and the paralysis is usually partial and incomplete. The pupil is more or less dilated, the reflex movement of the iris sluggish, accommodation is imperfect, and syndroma of Weber is frequently met with. This is paralysis of the motor oculi of one side, with hemiplegia of the opposite side. Other cranial nerves, such as the facial, auditory, olfactory, and trigeminal, may be affected. For symptoms, see "Cranial Nerves."

Owing to the affection of the arteries, hemiplegia and bulbar phenomena may accompany the symptoms above mentioned. Thrombosis occurring in diseased vessels, especially of the basilar or vertebral arteries, may produce an attack of apoplectic bulbar paralysis, in some cases quite sudden in origin. The writer showed a case at the Clinical Society of this kind, which occurred quite suddenly in a man six months after infection. The disease may take an acute or subacute course, it may extend over months or years, and show periods of exacerbations or remissions, but much will depend upon early and vigorous adoption of antisyphilitic treatment.

Meningitis and syphiloma on the brain convexity.—Some of the symptoms are independent of the situation of the disease, *e.g.* headache, often the first symptom, occurring months before other disturbances—vomiting, giddiness, stupor, and psychical disturbances. Then occur, often quite suddenly, violent circumscribed or general epileptiform convulsions, which follow one another rapidly. Besides the fits, other clinical symptoms may arise—monoplegia, or even hemiplegia, and frequently cortical speech disturbances of various kinds. Optic neuritis is by no means a common phenomenon in this brain affection. There is a predilection for the frontal and central convolutions to be the seat of the disease. The fits generally increase in severity, loss of consciousness supervenes, terminating sometimes in coma and death. By energetic treatment, however, these cases often make a good recovery.

Primary syphilitic arteritis.—Even without any meningeal affection, one of the earliest and most important symptoms present may be headache (usually more severe than that of arterio-sclerosis), giddiness, and drowsiness, and various psychical disturbances, not unlike some of those of dementia paralytica. Heubner asserts that headache, giddiness, sleeplessness, irritability, and weakness of memory often precede an apoplectic seizure. These symptoms, together with transitory aphasia, monoplegia, hemiplegia, hemianopsy, hemianæsthesia, and word-blindness (which may last a few minutes, a few hours, or a few days) are the expressions of a general and local disturbance of the cerebral circulation. As long as the lumen of the vessel is not obliterated by the formation of a thrombus, collateral circulation can restore the blood supply to the part; but, as the endarteritis proceeds, this becomes less possible, and the stagnant blood in the vessel clots, but gradually producing a progressive loss of function, paralysing successively the leg, arm, and face, or, *vice versâ*; sometimes quickly, producing hemiplegia and loss of consciousness, and resembling a true apoplectic fit. Sometimes there is hemiparesis, which passes on to complete hemiplegia, coma, and death. On the contrary, the patient may recover; but in the course of months or years the paralysis may return, or it may affect the other side, and the symptoms of pseudo-bulbar paralysis present themselves. In severe cases of multiple syphilitic arteritis with thrombosis, death follows in a short time, often with pyrexia, and the writer has seen several cases of thrombosis of the basilar and of both middle cerebrals die rapidly with a high temperature.

Multiple gummata.—The patient is demented, frequently maniacal or stuporose; he may have a peculiar speech disturbance, with tremors of the face muscles, and he does not respond to the calls of nature; pupils are unequal, and of sluggish reaction. Such cases closely resemble general paralysis; inasmuch as there is progressive dementia and paresis, but usually there is associated diplopia, ptosis, strabismus, optic neuritis, or some paralysis which points to a coarse lesion. Any one of these symptoms combined with severe headache would lead one to suspect multiple brain lesions of syphilitic origin, and in a number of cases which I have examined post-mortem, I have invariably found associated with the gummata, basic meningitis and arteritis.

Cerebro-spinal syphilitic meningitis.—To the cerebral symptoms described may be added spinal symptoms. Five cases the writer has lately seen showed the following symptoms:—marked psychical disturbance, mental confusion, loss of memory and delusions, vomiting, hiccough, severe headache, optic neuritis; while the spinal root symptoms were indicated by pain in the back, stiffness in the neck (and in one case opisthotonos), shooting pains, hyperæsthesia, and paralysis, with wasting of the muscles. One case recovered with treatment, the others died, and the diagnosis in each case was verified at the autopsy.

Finally, it must be remembered that syphilitic brain disease such as described in this chapter, may occasionally be accompanied, preceded, or followed by parasyphilitic diseases, such as tabes or general paralysis.

Diagnosis.—Attention to the above-mentioned types of cerebral syphilis, and the symptoms they manifest, both general and local, will enable a diagnosis to be made; but if possible, it should be confirmed by a history of syphilis. This is not always easy to obtain, but age, sex, and the life and habits of the individual will often lend valuable aid in forming an opinion. If it is impossible, and it often is, to find any specific evidence on the body, *e.g.* scar of chancre, enlarged glands, pigmented patches on the skin, old iritis, scars about the knees, swelling of the testicle, chorio-retinitis, etc., or history of abortions and still-born children in women, nevertheless it is often well to treat a doubtful case upon the assumption that it is of syphilitic origin. Large doses of iodide of potassium and mercurial inunction often clear up the diagnosis in a most satisfactory manner when other means fail. Seeing that syphilis may attack any part of the cerebro-spinal nervous system, causing general or local irritation or destruction, the complex of symptoms may come to resemble many diseases of the nervous system.

Prognosis depends upon a number of causes, and in syphilitic brain disease it is more hopeful than in other organic diseases of the brain. According to Fournier, one-third are cured, one-sixth die, and one-half improve to some extent; but, as a rule, severe symptoms remain in a large proportion, and the outlook is generally gloomy.

In syphilitic brain disease after 40, the prognosis is unfavourable. The influence of treatment is of special importance in the prognosis. If the disease is recognised in the early stages, before actual destruction of nervous tissue has been brought about, the prognosis is more hopeful.

Cases in which the clinical phenomena are related to cerebral irritation, due to a gumma upon the convexity or to pressure upon the cranial nerves, are more favourable than monoplegias, hemiplegias, and paraplegias, which are generally due to arteritis, with vascular occlusion by thrombosis, and subsequent softening; for although it is reasonable to suppose that resorption of the new tissue in the intima may take place (which we know happens in a gumma) under treatment, yet we are sure that no amount of iodide and mercury can lead to regeneration of nervous tissue in a necrosed area. If benefit does not occur within a week by energetic treatment with iodide, or in two or three weeks with mercurial treatment, the prognosis is unfavourable. Mixed cases of arteritis with gummata or meningitis are usually unfavourable. It is well known that some of the worst cases are those which have had a mild attack of syphilis, and possibly this may be due to inefficient treatment; but there is no doubt that cases which have not been treated properly are more liable to be followed by nervous disease. Symptoms of general arteritis are of particularly unfavourable omen; likewise those pointing to affection of the basilar artery.

Treatment.—The earlier a diagnosis is made, and the patient put under energetic treatment, the better. Probably the best method to administer the mercury is by inunction. One or two drms. of mercurial ointment may be rubbed into different parts of the body daily; the amount used must depend largely upon the effect it produces, for it is necessary to obtain tenderness of the gums, but to avoid salivation. Anæmic, ill-fed people are readily salivated. Hypodermic injections deep into the muscles, of solution of perchloride of mercury, $\frac{1}{16}$ gr. to $\frac{1}{8}$ gr., may be employed sometimes with advantage. Iodide of potassium in 20- to 60-gr. doses three times a day is, however, most valuable in syphilitic brain disease. Should the potassium salt not be tolerated by the patient, iodide of sodium may be employed. Perchloride of mercury, $\frac{1}{16}$ gr. to $\frac{1}{8}$ gr., may be administered with the iodide, if inunction is impracticable. It is very important to tell the patient to brush the teeth after each meal and to give a mouth-wash of chlorate of potash, otherwise the gums will be spongy before sufficient mercury has been absorbed to produce an affect. For the paralysis and contractures which become permanent, the same treatment may be adopted as in hemiplegia.

F. W. MOTT.

GENERAL PARALYSIS.

Syn., General Paresis; Dementia paralytica; Fr., *Paralysie générale;* Ger., *Allgemeine progressive Paralysie.*

Bayle was the first to describe this disease as a distinct morbid entity, 1826. It has been the study of numerous alienists in nearly all civilised countries. It is not uncommon for text-books of medicine to omit the description of the disease, but it is as truly an organic disease of the central nervous system as any other degenerative process, *e.g.* tabes, with which etiologically, pathologically, and in some respects clinically, it is closely associated.

The disease consists in a primary degeneration of the brain with secondary inflammation, associated with progressive dementia and motor paresis, almost invariably terminating fatally within a few years of the onset of symptoms.

Etiology.—General paralysis is a disease affecting urban populations rather than rural. It has increased rapidly of late years, and probably is one effect of syphilisation and civilisation. Fournier was the first to point out that general paralysis, like tabes, was due to acquired or inherited syphilis; he therefore terms it a parasyphilitic disease. The arguments which support this view are very strong, and in the writer's opinion conclusive as to syphilis being the most important factor in the production of the disease. He does not think, however, that it is proved that general paralysis may not occur in an individual who has no specific taint. General paralysis is a disease of large cities and towns, so is syphilis; men are affected much more often than women, and this applies to syphilis. If the disease affects women, they are of the lower classes, and it is very seldom that one meets with it in the well-to-do classes of women; but the highest in the land among males may be affected with general paralysis; this is true also of syphilis. The disease has been ascribed to alcohol, but in countries where intemperance is rife yet syphilis absent, general paralysis is unknown, *e.g.* rural districts of Ireland and Sweden. Against this argument it was formerly stated that in Egypt syphilis is very common, but general paralysis rare; it must, however, be remembered that it is only quite recently that asylums have been built in Egypt—most insane people being in Mohammedan countries looked upon as holy men; moreover, the recent reports by Warnock of the Cairo asylum show that 5 to 6 per cent. of the admissions are cases of general paralysis, and he considers that syphilis is the cause of the disease.

The writer has found a history of syphilis and signs of syphilis, such as scars and diseased vessels, much more frequently in general paralysis than in other classes of mental disease. Although general paralytics in the early stages of their disease frequently lead a most irregular life, yet the writer has never seen signs of recent syphilitic infection. Krafft-Ebing has shown that eight general paralytics, who exhibited no external signs of syphilis, possessed an immunity, for they could not be inoculated with the syphilitic virus.

One of the strongest arguments in favour of the view that syphilis is the factor of all others which causes general paralysis, is the existence of a juvenile form occurring in the subjects of congenital syphilis, thirty typical cases of which have come under my notice, with twenty-three post-mortem examinations. In more than 80 per cent. of these cases there was definite evidence of inherited syphilis. In 60 per cent. there were actual signs of congenital syphilis on the body. It is remarkable also, as supporting the syphilitic origin of the disease, that, unlike the adult form, the two sexes are affected with equal frequency. Other factors, such as worry, anxiety, mental strain and overwork, sexual excesses, alcoholism, blows on the head, sunstroke, and continual mental excitement, act as predisposing or exciting causes. Heredity plays a less important part in this disease than other forms of insanity. Those occupations which predispose to the etiological factors above-mentioned tend to the production of the disease. It affects especially men in the prime of life, in the thirties—the average age of death is 40. It is rare after 50; it may commence (excluding the juvenile form) even in the early twenties; but the most common period is between 30 and 40. The disease runs a slower course in women.

Morbid anatomy.—General paralysis is generally spoken of as meningo-encephalitis, as if the primary change were inflammatory overgrowth of the connective tissues, with secondary atrophy of the nervous tissue. With our modern conception of the "neuron" as an independent nervous unit, this theory is becoming untenable. Before, however, considering the minute changes, let us see what are the characteristic naked-eye phenomena. On removing the calvarium, the dura mater is often found thickened, but in nearly all cases it is noticeable that the dura mater is not fully distended in front. On slitting up the dura mater, in a typical case, a characteristic appearance presents itself in the obvious atrophy of the brain, especially of the frontal and central convolutions, with thickening and opacity of the pia arachnoid; and the sulci present an opalescent gelatinous-looking appearance, due to the cerebro-spinal fluid beneath the thickened membranes. On removing the brain, it will be noticed that there is a great excess of cerebro-spinal fluid. The weight of the organ may be diminished one third, in extreme cases; the wasting affects especially the cerebral hemispheres, and in the greater proportion of the cases there is a difference of weight, more often the left weighing less than the right. The lateral ventricles are greatly dilated, owing to the atrophy; and the ependyma of all the ventricles, especially the fourth, is thickened and granular. If now the membranes be stripped from the hemispheres, it will be observed that, especially in the frontal region, the brain substance tears away with the membranes, leaving a characteristic worm-eaten appearance. The grey matter of the cortex cerebri is diminished, and there is an absence or diminution of the normal striation from atrophy of the tangential and supraradial fibres. The atrophy also affects, but to a less degree, the spinal cord; and there may be obvious naked-eye systemic degeneration, the commonest form of which is similar to that of tabes, although, when examined microscopically, it is more frequently found that the pyramidal tracts are degenerated.

Hæmorrhagic pachymeningitis is not uncommon. This is caused by a vein giving way, by which a layer of blood, of a thickness usually proportional to the brain atrophy, occurs. If it is of recent origin, it looks like coagulated blood; but, owing to changes in the hæmoglobin, it gradually becomes of an orange-red colour; organisation of the clot occurs, and a regular laminated membrane exists, uniting the dura and pia arachnoid.

Microscopical examination of the brain by Nissl and Marchi-Pal methods exhibits the following changes. The former method shows a marked thickening of the pia arachnoid, and an overgrowth of the neuroglia tissue, at the expense of the nervous tissue. The regular layers of the nerve cells of the cortex are destroyed; the cells being in all stages of dissolution, from initial swelling with chromatolysis, to complete destruction, leaving only the nucleolus recognisable. Some cells are swollen up and no longer retain their pyramidal form; their processes are atrophied and appear broken off; others are almost globular, owing to swelling up of the nucleus, while again others present a shrivelled, atrophied appearance. The motor pyramidal cells do not present the normal stichochrome granules, and it is rarely that one finds a single healthy-looking cell in a section of the central convolutions. The spinal cord and other parts of the brain may show changes in the nerve cells, but they are not so widespread nor so advanced except in the tabetic cases. In fact, the anterior horn cells may sometimes have a normal appearance.

There is a great increase in the spider cells of the neuroglia, in those situations of the cortex where the atrophy of the nervous elements is most marked, namely, in the frontal and central convolutions and the island of Reil. It often appears as if all the basophile substance has passed out of the nerve cells into the glia cells, which are found in irregular groups particularly around the vessels. The vessels are especially observable, owing to dilatation of the perivascular lymphatics and cellular proliferation in the sheath; also numbers of mononuclear leucocytes are present; the blood contained in the vessels, however, does not contain excess of leucocytes as a rule. The glia cells are said to possess a phagocytic action (Bevan Lewis). By the Marchi and Marchi-Pal methods two important observations can be made, namely, absence of the tangential system of fibres, a most constant lesion, first described by Tuczek, and absence or great diminution of the super-radial and inter-radial association fibres, together with the existence of a variable number of degenerated fibres, in various stages of destruction. The writer considers the overgrowth of glia tissue and the local meningitis to be due to the irritation of the products of degeneration and venous stasis, for the following reasons:—Sections stained by Marchi method show that the cells around the vessels, owing to their phagocytic function, frequently contain the fatty product of degeneration. A substance, cholin, exists in the cerebro-spinal fluid in considerable quantities, and this, it is known, can arise from the breaking up of protagon into stearic and glycero-phosphoric acids and cholin. The writer has shown that the pia-arachnoid thickening corresponds very closely with the distribution of the veins opening into the longitudinal sinus. For obvious reasons this area would be more subject to stasis than areas which drain into the lateral sinuses. It should be mentioned, however, that many authorities consider the disease is a primary inflammatory meningo-encephalitis, with secondary atrophy of the nervous elements. Tabes, with which this disease is not infrequently associated, followed by, and in rare cases preceded, was formerly considered to be a primary sclerosis; it is now generally looked upon as a primary degeneration of the afferent system of neurons, with secondary overgrowth of the neuroglia.

General paralysis is a disease of impaired metabolism of the neurons. A cell nourishes itself, and is not nourished. The nerve cells, or, more correctly speaking, the neurons are perpetual elements, incapable of regeneration; consequently, if their durability is interfered with, they die early. The following facts support this argument. The brain does not waste in starvation—general paralytics often put on flesh during the second stage, and while rapid atrophic processes are going on in the brain. Examination of the blood has shown no evidence of noteworthy change in the corpuscles, although it is probable that it contains toxic products, the results of the degenerative process. Although there is in the last stage general emaciation, the organs do not, as a rule, show any important change except fatty degeneration of the parenchyma of organs or of the muscles. Fatty degeneration of the muscles is especially liable to occur if there have been prolonged epileptiform seizures. In prolonged cases reaching the third stage, the muscle fibres are greatly wasted and show no striation.

Symptoms.—The clinical history of general paralysis varies considerably. There are many types, but there are a few phenomena which are almost always present, namely—(1) progressive dementia, usually accompanied by mental exaltation or depression; (2) unequal, sometimes irregular pupils, reacting sluggishly or not at all to light, but reacting to accommodation; (3) progressive motor paresis, with tremor especially affecting the tongue and face muscles; (4) alteration of speech and writing; (5) altered knee-jerks, generally exaggerated, sometimes absent.

First stage.—After some precursory mental symptoms, brief or prolonged, but usually insufficiently manifest to attract attention, there is a noticeable change in the character and habits of the individual. He may become irritable and easily upset, violent or morose, taciturn and dull, previously having been of a genial, kind disposition. He may suffer with lapses of memory and intelligence, make mistakes in writing and spelling, and show a loss of sequence in mental processes. Previously tidy and clean in his person, he often becomes slovenly and careless. Sometimes a business or professional man, who has been accustomed to take life easily, develops an extraordinary activity; new and varied schemes enter his imagination for making his fortune or his reputation; but the successful issue of the scheme is only realisable by himself. These schemes lead him to squander his money in ambitious projects, and to pass whole days and nights in writing, calculating, composing, or talking, neglecting family and business, and not infrequently terminating in the bankruptcy court and ruin.

Another important sign is perversion of the moral and emotional faculties. A man who has led a blameless married life gives himself up to immorality, debauchery, and depravity; the sexual desire is often greatly increased; but the function being impaired, he often resorts to acts of indelicacy which may bring him into the police court. He may get into trouble by acts of theft or drunkenness, and this may lead to the discovery of his malady. Mental depression, hypochondriasis, and a tendency to suicide may be prodromal symptoms in some cases. There is, generally speaking, an enfeeblement of memory and loss of attention. Insomnia occurs usually during some period of the prodromal stage. There is also frequently loss of appetite and impaired digestion.

Second stage.—This period may be manifested by two mental states apparently opposed to one another. In one individual there is grandiose delirium, or delirium with excitement, acute mania; in another, hypochondriasis, or melancholia; but in both there is invariably a substratum of dementia, of intellectual enfeeblement, and incoherence. Delusions of health, wealth, strength, and grandeur are very frequent. They live in a palace of gold, are rulers of men, and possess millions and millions of money; yet sometimes, in reply to a question as to their occupation, they will answer correctly, plumber, painter, etc. They collect often stones, bits of paper, and other rubbish, which they tell you are priceless jewels. Possessors of untold treasures, they will often beg a pipe of tobacco; and although they are always going to do wonders, they are never surprised when they are told they have not begun. In contrast to this, there is a not unusual form of delusion, indicating mental depression, *e.g.* the wife and children are starving, he is being slowly poisoned, etc.; occasionally exaltation and depression may alternate.

Along with the early mental symptoms, generally after, but sometimes even before, special motor troubles appear. The alteration in speech is almost pathognomonic of the disease. There is at first a hesitancy, which may only be perceptible to practised ears, but in which there is no real fault of articulation once it is started; sometimes preparatory to and during the utterance, there is a tremulous motion of the muscles about the mouth. The hesitation increases, and instead of a steady flow of modulated articulate sounds, speech is broken up into a succession of irregular, jerky, syllabic fragments, without modulation, and often accompanied by a tremulous vibration of the voice. Syllables are dropped out, blurred, run into one another, or imperfectly uttered; especially is difficulty found with consonants, particularly the explosive lip sounds, b's, p's; again, linguals and dentals are difficult to pronounce. To ascertain the existence of a speech defect, various test words and sentences are used, *e.g.* preliminary, parallelogram, Mississippi, biblical criticism, the Irish artillery extinguished the conflagration. It is very seldom that a general paralytic can utter these correctly.

The same defect in writing occurs as in speech. The syllables and even the letters are disjointed. There is a tremor in the movement of the pen, syllables are left out, and sometimes words. The subject-matter of a letter may also form a valuable indication of the mental state. The ideation of a written or spoken word is based upon the association of the component syllables, the difficulty arises from an impairment of this faculty of association upon which spoken and written language depends—a function of the higher centres later acquired than the production of the articulate sound.

Motor paresis may be shown in the face, lips, and tongue; it does not amount to paralysis, as the patient can protrude the tongue; but it is often protruded by a succession of jerks, and generally is tremulous; the loss of expression in the face, which may even take on the appearance of a mask, is due to a loss of tonus in the muscles, by the absence of the emotions which serve as the natural stimulus in maintaining tonic contraction of the face muscles. On being told to show his teeth, there is often a fine tremor in the muscles raising the upper lip; a similar tremor may be observed in the extended fingers, and due, no doubt physiologically, to unequal innervation of the muscles. The muscles respond to faradic and galvanic currents, and do not give the reaction of degeneration. An important motor phenomenon is connected with the pupils. They may be large, medium-sized, or small; they are generally unequal, sometimes angular and irregular, and react either sluggishly or not at all to light, although generally they react to accommodation. This reflex rigidity to light is one of the most frequent physical changes met with in general paralysis. The Argyll Robertson pupil is a valuable diagnostic sign of this disease, as it is of tabes. Besides the iridoplegia, there may occur, as in tabes, transitory or permanent ocular paralysis. The knee-jerks are usually exaggerated, in some cases absent, generally indicating the tabetic form of the disease, occasionally present on one side, absent on the other. There is generally muscular weakness, affecting especially the finer volitional movements. In spite of the muscular weakness, the bodily weight may even increase during this stage.

With regard to sensory disturbances, it is difficult to estimate impairment of cutaneous sensation, although there is apparently blunting or even in some cases abolition of sense of pain; frequently they complain of neuralgias, attacks of migraine, and a feeling like the pressure produced by a tight-fitting hat upon the forehead. In cases of the tabetic type, grey optic atrophy is by no means uncommon. In 130 consecutive post-mortems made by the writer upon general paralytic cases, the optic nerves displayed naked-eye atrophy in ten; about 7 per cent., therefore, certainly have optic atrophy. As a rule, the fundus shows no other important change, except occasionally there is pigmentary choroiditis of syphilitic origin.

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Congestive apoplectiform or epileptiform attacks generally occur in the second and third periods of the disease; but they may occur before this. In some cases a fit may be the first symptom which leads to medical advice being obtained. The seizures may vary very much in their severity, *e.g.* simple congestive attacks are indicated by flushing of the face, restlessness, increase of tremors and twitchings, but not loss of consciousness; or the patient may suddenly have a fit of giddiness, or faintness, and fall down unconscious, or consciousness may be disturbed but not lost; there may be convulsive twitchings affecting one side of the body, or perhaps only the face, or a limb. Transitory hemiplegia or a monoplegia may follow, and if the right side is affected there may be a disturbance of speech and a transitory aphasia. The attack may last a few minutes to half an hour, and then the patient recover. The fits, however, usually recur; and sometimes the convulsions become very marked and even general, with complete loss of consciousness; occasionally there is tonic as well as clonic spasm. The unconsciousness may deepen to coma, and the patient die after a succession of convulsive seizures; yet as many as forty or fifty a day may occur and continue for several days, nevertheless the patient may recover. Usually there is permanent increase of the dementia following a bad attack, but not necessarily. Sometimes the disease runs its whole fatal course without any pseudo-apoplectic or epileptiform fits. In the pseudo-apoplectic attack, which is far less common than the epileptiform, the patient falls suddenly, becomes unconscious, the limbs relax, the breathing is stertorous, the face flushed, the temperature raised, and this condition may persist until death, or it may end in recovery. The temperature from the onset of the fit is usually raised two or three degrees; in this it differs from apoplexy, which is at first subnormal. In general paralysis, at other times than during seizures, the temperature is usually normal or subnormal.

In some cases maniacal paroxysms may occur; the patients shout and become violent, dangerous and destructive; while in others, the only psychical disturbance is progressive dementia; grandiose delusions are absent, and the patient becomes more incoherent day by day, losing all knowledge of time and place. Such people often exhibit no cortical irritation phenomena. If the patient does not die in a seizure, or from some complication such as pneumonia, he will pass into the third stage.

Third stage.—The patient now lives a vegetative existence; he sits with his head forward, his hands across his knees or in his pockets, often grinding his teeth, and indifferent to surroundings. The face is often dusky and flushed, the hands are cold and blue, indicating a feeble circulation; the skin has often an oily appearance. Indifferent to the calls of nature, he passes urine and fæces under him, so that he requires constant attention to prevent the formation of bedsores. Although he often eats enormously the minced food which it is necessary now to give him, to prevent him choking, yet he emaciates; not infrequently he satisfies his voracity by stealing the food of a neighbouring paralytic. Attendants in asylums regard this, from experience, as especially suggestive of general paralysis. The wasting and weakness progress, until the patient has to be put to bed, from which he seldom again gets up; death is frequently hastened by broncho-pneumonia or gangrene of the lung, owing to food entering the air passages; cystitis, with secondary pyelitis and infective nephritis; secondary infection from bedsores and ulcerative colitis. Should he, however, escape all these dangers (for, with the present care these patients receive in asylums, these complications are frequently avoided), the general paralytic may die so gradually from asthenia, that the writer has seen cases in which every breath for days might have been the last. The disease is essentially a brain disease—no important changes occur in other organs of the body except by complication.

Course.—In the galloping forms of general paralysis, the disease may only last a few months; for a patient with acute maniacal symptoms and restless delirium, refusing food and sleeping neither by day nor night, and with consequent rapid wasting of brain and body, dies very soon after the onset of symptoms; frequently the fatal termination is hastened by pneumonia, occasioned by exposure, owing to his tearing off his clothes continually. As a rule, the disease is fatal in two or three years; but the writer has seen cases which have become stationary and been discharged from the asylum, and a period of six to eighteen months may elapse before they return. It is not so rapidly fatal in women, and the opinion of the writer is that it is more rapidly fatal in town-bred people.

Diagnosis.—The diagnosis in the early stages of general paralysis is of the greatest importance, as ruinous consequences to the family or the individual may often be averted by timely interference. It is the early recognition of the disease that should be especially studied by the practitioner, for any layman can recognise a deranged mind when the disease is well pronounced. The early symptoms of the first stage may be mistaken for neurasthenia or alcoholism; but the age of the patient, the sex, the history of a change in the moral character of the individual, with loss of memory, the existence of unequal pupils, which are sluggish or inactive to light, but react to accommodation; slight hesitancy and alterations of speech, especially noticeable with the test words previously mentioned; facial tremors, together with altered knee-jerks, will generally serve to distinguish the disease from neurasthenia and from alcoholism; for although in the latter there may be dementia and even alteration of speech, the pupil phenomena are absent. The greatest difficulty, however, lies in those cases in which there is general paralysis combined with alcoholism: mistakes are thus often made by attributing the whole of the symptoms to alcohol poisoning, whereas it will be subsequently found that, when alcohol is withheld, the dementia and paresis progresses: in fact, it is sometimes quite impossible to state offhand whether a patient is suffering from alcoholic dementia or general paralysis, or from mania *a potu*, as distinguished from the mania of general paralysis combined with alcohol. The pupil phenomenon is the most reliable symptom, but sometimes it is absolutely essential to reserve decision until the effects of the alcohol have passed off, and it must be remembered that unequal pupils may be congenital.

Megalomania should not be mistaken for the grandiose delusions of general paralysis, for the latter is characterised by absurdity and want of system, associated always with a substratum of dementia.

Syphilitic brain disease, due to general endarteritis or multiple gummata, may be mistaken for general paralysis; the paralyses are, however, usually coarse, such as hemiplegia, monoplegia, aphasia, etc.—conditions which are rare in uncomplicated general paralysis. The syphilitic brain disease which most closely simulates general paralysis is multiple gummata: but in this there are signs of intracranial pressure and irritation, *e.g.*

headache, vomiting, and optic neuritis, associated with the dementia. Disseminated or insular sclerosis is distinguished by intentional tremors, more marked than that of general paralysis, nystagmus, and absence of the Argyll Robertson pupil; and the speech alteration is not the same in character, but slow and scanning; there may be also difficulty in swallowing, and there are frequently changes in the fundus. After 50, a form of progressive dementia with paresis, which closely resembles general paralysis, but which is due to general arterio-sclerosis of the cerebral vessels, may be, and often is, confused with true general paralysis. The apoplectiform fit of general paralysis resembles in some respects true apoplexy, but the latter seldom occurs at an age when the diagnosis of general paralysis is permissible, *e.g.* if a man between 30 and 40 is suddenly seized with an apoplectic seizure, it is probably either general paralysis, or thrombosis of a large vessel, due to syphilitic disease. The previous history of the patient will in most cases help to decide. In true apoplexy, moreover, the temperature is at first subnormal, whereas in general paralysis it is raised two or three degrees.

The epileptiform attack may be mistaken for Jacksonian epilepsy, of gummatous origin, and if the patient be seen for the first time, a history from the friends is the only help towards a decision. Uræmic convulsions should not be mistaken for a paralytic seizure, as the previous history and the existence of albumin in the urine and retinitis will enable a correct opinion to be formed.

Prognosis.—Essentially bad. General paralysis seldom lasts more than three or four years, sometimes only a few months. As previously said, the disease may sometimes undergo a remission; in most cases it would be safer, however, to assume that the diagnosis was wrong than that the case had been cured. Still, the earlier the patient comes under medical supervision, probably the better the prognosis as regards the duration, for if a patient be allowed to remain long in a state of restlessness, insomnia, and mental excitement, the disease is much more likely to progress rapidly. On the other hand, restraint from drink, sexual passion, and excitement generally, may lead to an abatement of the symptoms. The occurrence of numerous epileptiform or apoplectiform seizures is usually unfavourable, for they may end fatally, and such symptoms usually, but not necessarily, indicate a rapid course.

It is the writer's opinion that tabetic cases run a more prolonged course; and it is certainly a fact that seizures are less common in women than men, also that the disease is of longer duration, as a rule. The juvenile form of the disease differs in no essential points from that affecting adults. Grandiose delusions are, however, uncommon, and the epileptiform seizures are not so severe.

Treatment.—As soon as the disease is recognised, the patient should be put under such conditions as to ensure mental and bodily rest. Restlessness and insomnia should be treated by suitable drugs; sulphonal or trional in 15- to 20-gr. doses may be given repeatedly, or paraldehyde in 1-drm. doses; if these drugs do not act and there is acute mania, $\frac{1}{100}$ gr. of hyoscine may be given hypodermically; but it must be remembered that such drugs have a depressing action and must be used with care and discretion. Attention to the state of the bowels and the passage of the urine should be carefully observed; the diet must be regulated so as to place the patient under favourable conditions of nutrition. In the later stages of the disease the food must be minced, and if the patient be treated at home, he should have an attendant constantly with him. Mercury and iodide seldom do any good in this disease, but since they may have a most remarkable effect in pseudo-general paralysis of syphilitic origin, it is extremely important to differentiate these diseases, and if there be any doubt, to try large doses of iodide combined with mercurial inunction. The writer has seen some doubtful cases which have been saved by prompt anti-syphilitic treatment, and others which have died from the neglect of it. Care and attention should be given to prevent the various complications which have been mentioned above; otherwise there is little to be done for this incurable and hopeless disease.

F. W. MOTT.

DISSEMINATED CEREBRO-SPINAL SCLEROSIS.

Syn., Insular Sclerosis; Multiple Sclerosis; Fr., *Sclerose en plaques disseminées;* Ger., *Herdsclerose.*

A disease characterised by very varying symptoms, due to the formation of scattered islets of sclerosis in the brain, spinal cord, and cranial nerves.

Etiology.—This is very obscure. The disease is met with about equally in the two sexes, and nearly always occurs in the period between adolescence and middle life, being extremely rare in childhood and after fifty years of age. It usually attacks typically healthy young adults. Direct hereditary transmission is seldom met with, but a neurotic predisposition can usually be traced in the family. Many morbid influences favour the development of symptoms, notably grief, worry, and overwork. Not a few cases have followed acute specific fevers, such as typhoid fever and smallpox, and the sequence has been held to support the view that the disease was due to some toxine, possibly produced by a specific organism, but at present this is entirely hypothetical.

Pathology.—The disease is characterised by the presence of islets of sclerosis, scattered irregularly through the brain and spinal cord, and in some instances in the course of the cranial and spinal nerves. They vary in size from that of a small pea to that of a plum. Their naked-eye appearance varies at different stages. In advanced cases they have a warm grey gelatinous appearance, and may simulate gliomatous tumours. They are usually less firm than the surrounding tissues, so that the term sclerosis is a misnomer. In the early stage, however, the patches are often indistinguishable from the surrounding tissues in the fresh condition, and are only revealed after the cord has been placed in bichromate solution for a few days. This fact has no doubt helped to create the confusion between this disease and hysteria, many cases being supposed to be proved to be due to hysteria, owing to the apparent absence of a pathological lesion. The patches are found chiefly in the white matter. They occasionally invade the grey matter of the cord and medulla oblongata, but are hardly ever found in the cerebral cortex. In the cord they are usually small, but in the centrum ovale they may attain considerable size. They are most abundant in the cord, and in an advanced case are present at any level at which a section may be made, but each patch does not extend for a long distance vertically. The seat of election is in and around the tracts which conduct to and from the brain. Microscopic examination shows that where the process is early there is considerable dilatation of the vessels, with leucocyte infiltration around them, and increase of neuroglia, which separates the fibres from one another. In other words, there is an inflammatory condition, and not a primary degeneration. The medullated sheath disappears after a time, but the axon persists until a late stage. In consequence of this persistence of the axon, patches of considerable size may exist for a long time in the spinal cord without giving rise to the usual ascending and descending degenerations. Degeneration usually occurs first in the crossed pyramidal tract in the lateral columns.

Symptoms.—As might be expected from what has been said about the pathology of this disease, the symptoms vary very widely in different cases. It is usual to speak of three stages of the disease, the first being the period of development of symptoms, which varies in character and duration; the second, the period in which the characteristic symptoms are fully developed; and the third, the period of dissolution, when the organic functions fail, and the patient sinks from exhaustion, pneumonia, or septic kidney trouble. In the first period, or stage of invasion, the greatest variations are to be found. The cases fall into two clearly defined groups.

Typical or progressive type.—In this many or all of the following symptoms are present, not always in the same order. The patient complains of heaviness and loss of power in the lower limbs, and the deep reflexes are found to be exaggerated. Later, the legs become spastic, and ankle-clonus can be readily elicited. There is tremulous unsteadiness when the patient gets up from a chair and stands erect. When he tries to walk, his gait is unsteady as well as spastic, although the unsteadiness is rarely as marked as in well-marked cases of tabes, but in the early stages it may lead to uncertainty in diagnosis. In the lower limbs, weakness and spasticity are the prominent symptoms, while in the upper limbs unsteadiness predominates. In addition, nystagmus, occasional diplopia, and irritability of the bladder are usually early symptoms.

Intermittent or hysterical type.—This form has been very clearly separated and described by Buzzard. The cases are more frequent in females than in males. The onset of symptoms often corresponds with the occurrence of some mental or physical shock, such as a fright, the death of a parent, a love affair, or an accident. Following such an event, loss of power occurs in one or both lower limbs, sometimes only lasting for a short time, sometimes continued for weeks. There is often anæsthesia of the affected limbs, and not infrequently there may be convulsions of a functional type. There is also an alteration in manner, the patient becoming emotional and self-centred. After a time recovery takes place, often suddenly, and the patient only relapses after some fresh shock. The course of the symptoms, therefore, closely corresponds with those met with in uncomplicated hysteria, and the cases are often unrecognised as cases of organic disease until the second period, when the symptoms of disseminated sclerosis are unmistakable. But, on careful examination, even in these so-called hysterical cases, indications of the presence of organic disease can usually be found. Of these the most important are—nystagmus, or lateral deviation of the eyeballs, occasional diplopia, pallor of the optic discs, usually unequal on the two sides, and over-excitability of the bladder, the first call having to be attended to immediately—precipitate micturition. In the second stage the symptoms are well developed. They are not always all present or in equal degree. In one case the tremors will be the most striking feature, in another the weakness, in another spasticity, and so on. The symptoms undergo steady aggravation, until the patient passes into the third stage, in which, as he becomes more helpless, the tremors subside, intelligence often becomes clouded, and, the sphincters becoming paralysed, evacuations are passed under him. Death usually occurs from exhaustion, from discharging bedsores, or from septic kidney disease, secondary to septic cystitis; or pneumonia, bulbar palsy, or some intercurrent malady may carry off the patient.

Considering the symptoms in detail, the following are the most prominent:—

Tremors.—The tremors in disseminated sclerosis are usually spoken of as intention tremors, that is, the tremors are only obvious when some voluntary movement is made. They almost always cease when the patient is at rest. They are usually seen first in the upper limbs, but later on affect the legs, the head and neck, and the trunk. On any voluntary movement (such as slowly touching the tip of the nose with the tip of the index finger), rhythmical movements of the arm are excited, which go on increasing in amplitude while the effort is kept up. This is well seen if the patient is made to hold a tumbler which is nearly full of water. At first the tremors only cause a rippling of the water, but they soon become more and more violent until the water is spilt. As soon as the arms are at rest the tremors cease. Any emotional excitement aggravates them. They tend to disappear as the limbs become weaker. In severe instances they are so violent as to make it impossible for the patient to leave his bed, although he may not be paralysed, as the effort to sit upright brings on such violent movements of the trunk and other muscles concerned that the patient would be jerked out of a chair.

The tremors differ from those of paralysis agitans in being more irregular, both in time and extent, and in ceasing when the patient is at rest in bed. In paralysis agitans one limb is usually affected considerably before the one on the other side, and the head and neck are rarely involved. The movements differ from those of chorea, in that they only follow the particular movement in which the limb is engaged, and do not spread to those of other joints. Similar intention tremors are also met with in some cases of mercurial poisoning, and in rare cases of intracranial tumour, especially in children.

Paralytic symptoms.—These usually occur first, and are most marked in the lower limbs. They nearly always affect the limb as a whole, and it is only in exceptional cases that there is atrophy of some muscle or group of muscles, indicating that a patch of sclerosis has attacked a corresponding area in the anterior cornu of the cord. A characteristic feature about the paralysis of the legs is its variability from time to time. Often in the early stages there is sudden momentary loss of power, the patient stumbling or actually falling, but recovering himself almost immediately. Such transient loss of power is rarely met with in the arms. In later stages, the loss of power, although subject to variations, progresses. In the legs, loss of power is accompanied almost always with spasticity. This spastic condition is subject to sudden increase, the legs being suddenly thrown into a rigid extended position, in which he may slip from a chair. Although this rigidity is suddenly induced, it takes some time, often several hours, for it to pass off. It is usually attended with much pain of a cramping character. Loss of power becomes complete ultimately in the legs, but there is almost always some power remaining at the last in the arms.

Sensory symptoms.—These are less marked than the motor. There is usually slight diminution of sensibility to touch and pain in the lower limbs, which varies greatly from day to day, and is rarely complete. Subjective sensations are usually pronounced, taking the form of "deadness," "pins and needles," a feeling that the leg does not belong to the patient, etc. Occasionally there is great pain in the limbs, often associated with cramp. In rare cases there are vasomotor and trophic changes in the feet, which are swollen, purple, and extremely tender (erythro-melalgia).

Tendon responses.—There is almost always great exaggeration of the knee-jerk, and, after the early stage, ankle-clonus can be elicited. The elbow-jerk is usually exaggerated, and the jaw-jerk can often be obtained. In rare instances the knee-jerks are lost, probably from involvement of the reflex arc by a patch of sclerosis.

Organic reflexes.—Over-excitability of the bladder, requiring the patient to attend to the first call, is usually one of the earliest symptoms of the disease. In the second stage there is occasional difficulty in commencing to micturate, and later on there may be retention, which usually leads to incontinence. In the latter case there is generally a considerable quantity of residual urine, which is specially apt to decompose, and lead to septic cystitis, particularly in females. Unless the greatest care is taken, the septic inflammation is apt to spread up the ureters and to affect the kidney, ultimately causing death. Incontinence of fæces sometimes occurs as a late symptom, but is rare in the early stage. Swallowing is rarely interfered with till late in the disease, and then only if the sclerosis invades the medulla oblongata.

Affection of speech.—Speech is often affected in disseminated sclerosis, but by no means as often or as early as is usually represented, and frequently remains unaltered throughout the whole course of the disease. The voice when affected is somewhat husky and monotonous, and the component syllables are spaced out and uttered with deliberation and effort, all the syllables being accented thus—ar-til-le-ry (syllabic or staccato speech). If the patient tries to speak rapidly, he slurs the words, and the speech may then be suggestive of general paralysis. A syllabic slurring speech is also seen in Friedreich's hereditary ataxy, and in some hereditary cases of lateral sclerosis.

Special senses.—Hearing, taste, and vision are rarely interfered with, but disturbances of vision are common in the course of the disease. At first the disturbance consists in more or less sudden failure of vision in one eye, which may entirely pass off the next day. In its characters it closely resembles hysterical amblyopia, for which it is often mistaken. Later, there is considerable loss of visual acuity. The patient can only read large print in a good light, and finds it increasingly difficult to recognise faces. The loss of vision is usually progressive, although complete blindness is rarely if ever met with. Ophthalmoscopic changes are met with in 50 per cent. of the cases, varying in degree from slight pallor to marked white atrophy, but are rarely an accurate measure of the functional loss of vision. The change is rarely equally advanced in the two eyes, and differs from the similar conditions of the disc in tabes, in the much slighter corresponding loss of vision. There may be advanced pallor, with diminution in size of the vessels, and yet the patient may be able to read the paper comfortably. On the other hand, there may be great diminution of vision with little ophthalmoscopic change. The presence of atrophy, although of great importance from a diagnostic point of view, should not mislead a medical man into giving a hopeless prognosis as to vision.

The fields of vision are usually uniformly contracted both for white and for colours, the shape of the fields being similar to that of the fields in cases of hysteria.

Nystagmus is usually an early and persistent symptom. There may be every gradation, from a little jerking, when the eyes are turned laterally to their fullest extent, to constant nystagmus in all positions.

Diplopia is usually an early but intermittent symptom. It depends on transient weakness of one or other of the ocular muscles. Permanent strabismus is rare.

Mental change.—There is no characteristic mental change associated with the disease, but mental peculiarities are often met with among the subjects. In the initial stage, there is frequently so much emotional disturbance that the signs of organic disease are overshadowed. When the disease is established, the patients are usually optimistic, and have a firm expectation of recovery. As the disease progresses, the mind may become more or less enfeebled.

Diagnosis.—Owing to the fact that the patches of sclerosis may affect any part of the cord, and many parts of the brain, the symptoms may resemble diseases usually associated with morbid changes in a particular region or tract. Thus, if the first patches are in the posterior column of the spinal cord, the symptoms may simulate those of tabes; if in the lateral columns, those of lateral sclerosis; and if in the medulla oblongata, those of bulbar paralysis. As, however, the lesions in disseminated sclerosis are usually multiple, there is as a rule not much risk of a mistake, if the examination is thorough. The inco-ordination, weakness, and spasticity, coupled with nystagmus, are very suggestive of the presence of a cerebellar tumour. In most cases, however, headache, vomiting, and double optic neuritis are prominent symptoms in cases of cerebellar tumour, while they are hardly ever met with as a symptom of disseminated sclerosis. The disease with which it is most likely to be confounded is hysteria. The remissions in the early stages, the frequency with which symptoms are brought on by emotion, etc., and the frequent occurrence of transient amblyopia, are very suggestive of hysteria, which is often confirmed by the emotional behaviour of the patient. Careful search has to be made for the signs of organic disease, as described in the section on Symptomatology. It should be a rule never to make a diagnosis of hysterical paralysis until symptoms of disseminated sclerosis have been searched for and excluded.

Prognosis.—Although it is characteristic of the disease to exhibit marked remissions, often lasting for months in the early stage of the disease, there is on the whole a loss of ground, and permanent recovery does not occur. Death takes place from some intercurrent malady, or from septic cystitis and pyelitis, or occasionally from involvement of the vital centres in the medulla oblongata.

Treatment.—No treatment by drugs appears to have any effect in checking the course of the disease. Rest in bed for a few weeks often has more effect in diminishing the tremors than any other treatment. Electrical treatment sometimes gives the patient comfort, but does not appear to influence the disease. Hyoscine, physostigma, and many other drugs have been tried, but the benefit obtained is not conspicuous. The patient must be guarded against fatigue, excitement, and worry, which have a strikingly prejudicial effect on the course of the disease. Care must be taken to see that the bladder is thoroughly emptied, as retention with subsequent paralysis is apt to occur. If a catheter has to be passed, careful antiseptic precautions must be taken, as cystitis is very apt to be set up, and tends to travel upward and give rise to septic inflammation of the kidney. When cystitis is present, the bladder must be washed out daily with some non-irritating antiseptic, such as a warm saturated solution of boracic acid.

W. S. COLMAN.

BULBAR PARALYSES.

A number of different morbid conditions may give rise to a group of symptoms, the principal features of which are paralyses of the muscles concerned in speech, swallowing, phonation, and mastication, or of the eye muscles. It is essential, therefore, to differentiate the various causes of these affections. Reference to fig. 18, p. 662, will help the reader to understand the groups, which will be considered in fuller detail under separate headings.

It is well to remember that the nuclei giving origin to the motor cranial nerves are homologues of the spinal motor nuclei in the anterior horns, and that they are often simultaneously affected. Every striped muscle is under the control of two sets of neurones—a cortico-spinal and a spinal motor; the former, as the diagram shows, proceeds from some definite area of the Rolandic region, conveying impulses controlling the action of lower motor neurones. Paralyses then may arise in the muscles, innervated by the motor neurones of the bulb—(1) by destruction of the cortical neurones or interruption of their axon fibres in their course to the bulbar nuclei; the interruption, however, must be bilateral, because from association the representative movements can be voluntarily controlled by one hemisphere; (2) by destruction of the motor nuclei in the bulb, or of their nerve fibres in their course from their cells of origin to the respective muscles, which they innervate. An important distinction between these two groups is obviously afforded by the condition of the muscles themselves and their reflex excitability; in the former, the muscles will not be atrophied, except from disuse, and the electrical reactions will be normal; in the latter, the muscles will be atrophied and give more or less the reaction of degeneration, and the reflexes will be abolished. Often the two forms are more or less combined. We can classify these affections anatomically into three great groups of (1) affection of the cortico-bulbar neurones, (2) affection of the bulbar neurones, (3) mixed forms. Or we can classify them, pathologically, into primary and secondary degenerative processes. The former are characterised by their insidious onset, progressive character, and the successive affection of functionally correlated groups of neurones presiding over particular groups of muscles subserving special functions, and often associated with a corresponding change in the homologous structures of the mesencephalon and spinal cord. In all these primary degenerations it is probable that both the upper and lower systems of neurones of the motor efferent path are simultaneously, if unequally, affected. In the second group we have lesions involving the nuclei of the motor cranial nerves and adjacent structures; they are usually sudden in onset, and are caused by inflammatory processes, or are secondary to vascular change leading to hæmorrhage or thrombosis. There are, besides, two somewhat rare conditions, in which the nuclei themselves are not involved in any morbid process recognisable by our present means. They will be treated of under Pseudo-bulbar Paralysis and Bulbar Paralysis without anatomical change—myasthenia gravis.

POLIO-ENCEPHALITIS SUPERIOR ACUTA.

The clinical symptoms of this disease were first described by Wernicke. The disease is not common, the writer has only seen one case of it. Chronic alcoholism is the usual cause; it has, however, been a sequel of infectious diseases, especially influenza, and it may arise from toxic substances contained in unsound meat, sausages, and fish; probably these cases are due to the poison engendered by the *Bacillus botulinus.* It occurred also in a case of sulphuric acid poisoning.

It has been shown that the symptoms are due to an acute hæmorrhagic inflammation of the grey matter in the floor of the third ventricle, the aqueduct of Sylvius, and sometimes extending to the floor of the fourth ventricle, causing damage to or destruction of the nuclei of the cranial nerves taking origin in those regions.

The onset is acute headache, giddiness, vomiting, usually, but not necessarily, preceding delirium or some form of disturbance of consciousness, such as drowsy restlessness, or less frequently, stupor and apathy; sometimes there is stiffness in the neck. Associated with one of the above mentioned conditions are paralytic affections of the eye muscles: usually all the muscles are affected, but single groups may escape, *e.g.* the levator of the eyelid and the sphincter of the iris and the ciliary muscle. Nystagmus is not uncommon, and not infrequently there is an oscillating gait simulating cerebellar ataxy; furthermore, there may be tremor and paresis in the limbs. The articulation is affected, and there is usually some facial paresis which may be unilateral or bilateral. The tendon reflexes are either normal or increased. Fever is uncommon, the temperature being generally normal or subnormal. The pulse is accelerated, and towards the end of life it is small and frequent; the respirations are hurried and shallow, and the disease runs an acute or subacute course, terminating fatally, as a rule, in from eight to fourteen days; recovery does, however, sometimes take place.

POLIO-ENCEPHALITIS INFERIOR ACUTA AND SUBACUTA.

An acute or subacute inflammation of the bulb, attended by the sudden development of the complex of symptoms known as bulbar paralysis. It is an inflammatory process, analogous to polio-myelitis or polio-encephalitis. The condition was first described by Leyden as bulbar myelitis. The inflammatory process may extend from the third ventricle to the calamus scriptorius, resulting in a complex of symptoms of ophthalmoplegia and bulbar paralysis. Sometimes polio-encephalitis and polio-myelitis may occur combined, causing in addition to the above, symptoms of affection of the motor cranial nerves, diffuse or circumscribed atrophic spinal paralysis.

ACUTE APOPLECTIC BULBAR PARALYSIS.

Softening or hæmorrhage are the commonest causes of acute bulbar paralysis. In persons under 40, it may be caused by embolism, but is usually due to syphilitic endarteritis of the basilar or vertebral arteries, with thrombotic occlusion of one or more of the terminal branches supplying the nuclei in the floor of the fourth ventricle. In older persons it is more often due to arterio-sclerosis or atheroma. The former condition is associated with the existence of miliary aneurysms, one of which may rupture, and the latter may give rise to softening, owing to stasis and coagulation of the blood in diseased terminal arteries. Various phenomena may precede the onset of the bulbar symptoms as the result of the arterial disease, namely, sleeplessness and headache, especially of the occipital region and neck, and sudden giddiness or loss of consciousness. The giddiness may be so severe as to cause the patient to fall down, and it may be accompanied by vomiting, but rarely by convulsions. Bulbar paralysis ensues, seldom taking more than two days to reach its full development, and causing dysphagia and dysarthria, or even complete loss of speech. At the onset there may be a condition of trismus, caused by tonic contraction of the muscles of mastication, this, however, being followed by loss of power. There is generally a symmetrical paralysis of the lower facial muscles and paralysis of the tongue, the soft palate, and the pharynx. The laryngeal muscles may be paralysed on one or both sides; the adductors are especially prone to be affected, causing hoarseness or aphonia. Sometimes the bulbar paralysis is accompanied by paralysis of the limbs, with rigidity and increase of the deep reflexes on one or both sides. As a rule, there is not much muscular atrophy, for the paralysis is due more to interruption of the fibres of the cortico-bulbar system on their way to the nerve nuclei, than to destruction of the nuclei themselves. The facial expression suggests weak-mindedness, as the most trifling circumstance causes emotional mobility of the facial muscles with the expression of weeping, less often laughing. Sensory phenomena are, as a rule, not prominent symptoms, but there may be paræsthesia, hyperæsthesia, hemianæsthesia, or pains in one side of the body, or anæsthesia over the distribution of the fifth nerve on one side. Dyspnœa and Cheyne-Stokes breathing may come on, and are of fatal omen; as also are acceleration of the pulse and high fever. The course of the disease depends upon the severity of the phenomena and the cause. Syphilitic cases are the most favourable, because collateral circulation can be restored before extensive necrobiosis has occurred. A patient under my care told me that 6 months after contracting syphilis, while seated at the piano, he felt faint; he got up to take a glass of water, and found he could not swallow it, the fluid regurgitating through his nose; he had lost his speech, and was unable to tell his friends what was the matter. A few days later, in addition to the bulbar paralysis, hemiplegia of the right side and weakness of the left developed. A partial but still satisfactory recovery occurred with vigorous antisyphilitic treatment. Hæmorrhage into the pons is nearly always fatal, so also is thrombosis of the main trunk of the basilar. Not so serious is thrombosis of one vertebral.

Bulbar paralysis may arise in the course of acute multiple neuritis. I have myself twice found degeneration of the vagus nerves in alcoholic polyneuritis. Again, severe forms of bulbar paralysis have been described by Eisenlohr and Müller in leukæmia, probably due to blocking of the minute capillary vessels by the myelocytes, or to multiple hæmorrhages. Bulbar paralysis may also be brought about by the compression of tumours growing from the base of the skull or the cerebellum, by tumours in the pons and medulla, by gummatous meningitis, and by the compression of an aneurysm of the basilar or vertebral arteries.

PSEUDO-BULBAR PARALYSIS AND CEREBRO-BULBAR PARALYSIS.

Pseudo-bulbar paralysis, as its name implies, is a disease in which the symptoms are like those of bulbar paralysis, but the lesion is not situated in the bulb. It is due to hæmorrhage or softening in both hemispheres, causing bilateral interruption of the path of voluntary impulses from the lower portion of the Rolandic cortex to the motor nuclei of the bulb. It is usually associated with bilateral weakness or paralysis of the limbs, sometimes with hemiplegia of one side and hemiparesis of the other. It develops as a result of successive apoplectic attacks, affecting first one side of the body and then the other. Emotional disturbance, with a tendency to laugh or cry, is an especial feature of the disease; and since it frequently arises from multiple lesions of the hemispheres, dementia in a variable degree is usually present, also apathy and confusion of ideas. The muscles are not wasted, except from disuse, reflexes are present, and there is no reaction of degeneration. Severe disturbances of respiration, and even of phonation, are usually absent. The writer has seen two cases arising from multiple bilateral softenings in the basal ganglia and internal capsule.

Cerebro-bulbar paralysis is commoner than the former. The vascular lesions are not limited to the hemispheres, but also affect the pons and medulla. Along with atheroma of the larger arteries, microscopical examination reveals a general arterio-sclerosis cerebri, with macroscopic or microscopic hæmorrhages, cysts, or softenings. The symptoms depend upon the extent, size, and distribution of these lesions. The arterial disease being a progressive degenerative process, the symptoms are progressive. There is usually some disturbance of the psychical functions, such as irritability of temper, confusion of ideas, hallucinations, delusions, and illusions; and there is difficulty in swallowing and speech, with a spastic paresis or paralysis of the limbs, which increases with each successive apoplectic attack; the patient finally becomes quite helpless and bedridden, hardly able to move, swallow, or speak, and liable to severe attacks of dyspnœa and Cheyne-Stokes breathing, which paroxysms may last several hours. Quickening of the pulse and arrythmia may also occur. Marked anæsthesia is seldom present, and there may or may not be control over the sphincters.

The prognosis in most cases is unfavourable, but I have seen patients so affected live years before death from some fatal complication has occurred. The treatment of acute bulbar paralysis depends upon the cause. In those conditions which correspond to polio-myelitis the treatment is similar; in other conditions, careful examination should be made for Bright's disease and valvular disease of the heart, and in their absence, even if there is no history or evidence of syphilis, mercurial inunction and large doses of potassium iodide should be tried without delay. The great difficulty in these cases is the feeding, and it may be necessary and advisable to feed the patient and administer the medicine by means of a soft tube passed through the nose. Later, in nuclear and infranuclear cases, if the muscles are atrophied, galvanism, combined with the administration of strychnine, may be tried.

PROGRESSIVE BULBAR PARALYSIS.

Syn., Glosso-pharyngo-labial Palsy, Duchenne.

This disease was first described by Duménil in 1859, but the classical description of Duchenne in the following year first brought about its recognition as a distinct disease. The morbid microscopical anatomy was first described by Charcot and Leyden.

Etiology.—It is a rare disease, affects both sexes pretty equally, comes on in the fifth or sixth decades of life, and is rarely met with under 40. The causes are unknown; exposure to cold, shock, mental trouble and worry, injury and stress, have been assigned as causes. It is frequently associated with progressive muscular atrophy.

Pathology.—To the naked eye little can be seen, but microscopically the various motor nuclei of the hypoglossal and facial, glosso-pharyngeal, vagus, accessorius and motor of the fifth show characteristic changes similar to those of progressive muscular atrophy. The nerve fibre network is atrophied, and the ganglion cells have either disappeared or are in all stages of degeneration; their processes are broken off, the cell bodies shrunken or only represented by a little clump of pigment, the Nissl granules absent, and but few cells are left in an advanced case. The motor nerves, which are the axis cylinder projections of these cells, and form the intra- and extra-bulbar roots of the corresponding cranial nerves, show a naked eye atrophy. Naturally, the nerve fibres in the muscles are degenerated; the muscle fibres themselves are either in a state of fatty degeneration or only show the sarcolemma sheath, and often fat replaces the atrophied fibres. The normal striation is seen in but few fibres. The muscle fibres that are undergoing degeneration show usually a proliferation of the nuclei of the sarcolemma.

Symptoms.—The onset is insidious, and the first noticeable symptom is slight indistinctness of speech, due to imperfect articulation of certain sounds in which the tongue is engaged, for example, the production of the consonants *l*, *r*, *n*, *t*, which require the elevation of the tip of the tongue to the palate. This imperfection in speech becomes more obvious when the patient is fatigued. Later, in consequence of the affection of the lips, such letters as *o*, *u*, *p*, *b*, *m* are indistinctly pronounced. Simultaneously, or later, owing to weakness of the soft palate, a nasal character or an increased difficulty in speech arises. Soon, associated with the dysarthria, there is a difficulty in swallowing, partly due to the tongue being unable to convey the food to the back of the mouth, partly because the tongue cannot assist in bringing the food back between the teeth, and it accumulates between the lips and the gums, partly because the pharyngeal muscles are no longer able to seize the food and start the reflex process of swallowing, and partly because the soft palate, being paralysed, does not effectually close the nasal passages, so that, especially with fluid nourishment, regurgitation through the nostrils may occur. As the disease proceeds, the difficulty in speech and swallowing is increased by the affection of the laryngeal muscles, the pitch of the voice is lowered, owing to paralysis of the adductors, the glottis is imperfectly closed during deglutition, and consequently there is a tendency for fluids, and even food, to pass into the larynx and set up fits of coughing, which, however, are ineffectual to expel the particles, because the glottis cannot be closed and the expiratory blast is of insufficient force. For the same reason, such sounds as can be uttered are feeble, hoarse, and indistinct. Later, even the muscles of mastication may be affected, and the disease, spreading into the respiratory centre, gives rise to attacks of dyspnœa. The intellectual faculties are, as a rule, unimpaired, although the facial expression, and the curious emotional mobility with the tendency of the patient to burst into tears or laughter, would suggest weak-mindedness. The patient presents a characteristic appearance: the mouth half open, the tongue lying motionless on the floor of the mouth, the tip behind the dental arch, unable to speak, and unable to swallow the saliva which is continually flowing out of the angles of the mouth, and, as Charcot has graphically portrayed, a slate in one hand to express his wants, and a handkerchief in the other to mop up the viscid saliva which is continually dribbling from the angles of the mouth. While the lower half of the face is so strikingly affected, the upper half retains its normal expression and power of movement. There is no loss of sensibility, but the pharyngeal, palatal, faucial, and laryngeal reflexes are lost, owing to the affection of the motor path of the reflex arc. The muscles are atrophied, especially the tongue, which cannot be protruded in the later stages even a short distance beyond the teeth; it often appears broad and flabby, this, however, being due to fat and fibrous tissue replacing the muscle; generally, however, it is atrophied, and its surface wrinkled and furrowed. Fine fibrillary twitchings of the tongue, less often of the lips, can be seen. The lips are thin, but sometimes retain their normal thickness. Electrical reactions are for a long time normal, but when a sufficient number of muscular fibrils are affected, a partial reaction of degeneration can be obtained.

The course of the disease is always progressive, although sometimes apparently the degenerative process halts in its progress and gives false hopes; but the disease seldom lasts more than a year or two. The causes of death are septic broncho-pneumonia, excited by the entrance of food into the air passages; failure of respiration, owing to involvement of the centre in the medulla, or weakness of the respiratory muscles; or tachycardia and heart failure, due to affection of the cardio-inhibitory centre. Cases have been known in which the frequency of the pulse towards the close of life has reached 140–160.

Diagnosis.—This is given on page 764.

Treatment.—Being an incurable disease, little can be hoped for. Tonics, such as arsenic and strychnine, have been given. Gowers recommends hypodermic injection of strychnine combined with $\frac{1}{20}$ gr. morphine. Very small doses of atropine might be added to check the excessive flow of saliva. Electricity has been tried, but with unsatisfactory results. The only thing usually to be done is to take care that the patient is fed in such a way as to avoid food passing into the larynx. Fluid nourishment is often more dangerous than very soft pulpy food, so that a minced diet is the most suitable. But it may be essential to feed the patient by a soft tube, and great care must be taken in its passage, as the larynx is insensitive.

It will be more convenient to consider chronic progressive ophthalmoplegia at the end of this article.

BULBAR PARALYSIS WITHOUT ANATOMICAL CHANGE.

Syn., Myasthenia Gravis, Asthenic Bulbar Paralysis, Asthenic Paralysis.

The first record of this disease was by Wilks, who describes in the Guy's Hospital Reports of 1877 a case of bulbar paralysis in which no anatomical changes were found post-mortem. In the following year Erb described a case as a peculiar form of bulbar disease. These observations did not attract much attention for ten years, when Oppenheim and Eisenlöhr described cases; and now some sixty cases have been put on record. Little attention had, however, been devoted to the disease in this country until quite recently, although for some time past it has had a recognised place in German text-books of neurology. An important contribution to this subject has lately been made by Buzzard and a valuable critical digest by Campbell and Bramwell appeared quite recently.

Etiology.—The disease affects both sexes equally. It occurs as a rule, however, at an earlier age in women. The youngest case on record was 12, the oldest 55; it cannot be said that occupation has any definite relationship, although manual workers are said to be more liable to it. The patients sometimes have previously enjoyed good health, but when women are affected, they are usually anæmic. An attack of an acute specific fever has preceded the onset of symptoms in some cases. Cold, over-exertion, and other conditions, are said to have been exciting causes in individual cases. Syphilis and alcohol appear to play no part in this disease.

Pathology.—The state of our present knowledge does not allow us to make any definite statement as to the nature of this disease; it is probably a toxic paralysis of some part of the voluntary neuromotor mechanism.

The myasthenic reaction would support the view that the poison affected the neurone rather than the muscles, and very probably at its most vulnerable point—the motor end-plate. The changes which have been observed in the motor neurones by Nissl's method are too inconstant and variable to help in the elucidation of this problem. It is conceivable that some toxic fatigue products are imperfectly dealt with, and poison the motor end-plates.

Symptoms.—The onset is usually gradual, and the characteristic weakness shows itself first as a rule in the muscles supplied by the bulb. Ptosis, usually bilateral, and often more marked on one side than another, is an early symptom; owing to palsy of the associated occipito-frontales, the forehead is not wrinkled, and the only way in which the patient can therefore look in front of him is by throwing the head back, but then often, on account of the weakness of the neck muscles, he is unable to do this. Weakness of the external muscles of the eyeball with diplopia is a frequent symptom; and a striking feature of the diplopia is an alteration of the relative position of the two images at different times. Pupil changes are quite exceptional. Difficulty in mastication is one of the most constant symptoms of the disease; sometimes there is dribbling of saliva from the mouth like a case of true bulbar paralysis. The immobile, expressionless face, and the drooping upper eyelids with inability to wrinkle the forehead, to raise the eyebrows, or to frown, is especially characteristic of this disease.

Difficulty in swallowing is another symptom which is seldom absent, and is very important, because in several instances death has followed attacks of choking brought on while swallowing. Weakness of the soft palate rendering the voice nasal and causing regurgitation of fluids through the nose, is common, but paralysis of the laryngeal muscles is of less frequent occurrence. The muscles of the throat and limbs may be affected, and alarming, even fatal, attacks of dyspnœa may come on from affection of the respiratory muscles. The occurrence of fatigue on any voluntary exertion is especially characteristic, and the speedy production of muscular exhaustion constitutes the most striking feature of the disease. Sometimes the drooping of the eyelids and the other evidences of paresis are only noticeable towards the end of the day. Persistent voluntary exercise of the affected muscles, even in the early morning after sleep, causes the characteristic exhaustion and paralysis, and very significant of the disease are the variations in the intensity of the symptoms and the occurrence of remissions. In 1895, Jolly described the characteristic electrical reaction of the muscles which is termed the myasthenic reaction.[1]

If the muscle is excited by the faradic current, the electrode being kept in contact with the muscle, the contraction becomes gradually feebler until finally the muscle ceases to respond. If, however, the electrodes be removed, and, after a few minutes, reapplied, contraction is obtained. The muscles, however, cannot be exhausted by galvanic stimulation. There is no muscular atrophy. The knee-jerks are rather more active than normal; there are no sensory symptoms, and muscular sense and co-ordination are not affected.

Diagnosis.—The diseases most liable to be confounded with this affection are bulbar paralysis and a myotrophic lateral sclerosis, polio-encephalitis, muscular dystrophies, especially Landouzy-Déjerine paralysis, hysteria, and a form of Landry's paralysis affecting the muscles innervated by the medulla. The most important points to bear in mind are the absence of muscular atrophy, normal electrical excitability or the myasthenic reaction, considerable remissions and phenomena of fatigue on exertion, together with the characteristic affection of muscles supplied by the bulb.

Prognosis and treatment.—This is very serious, more than a third

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[1] *Differential Diagnosis of Bulbar Paralyses.*

I. Acute Bulbar Disease.	TRAUMATISM . . .	History of a blow or fall; condition in which patient is found. Possible evidence of disease of cervical vertebræ, involving atlas and axis, fracture or dislocation being brought about by a trivial strain or blow.
	HÆMORRHAGE . . .	Sudden onset during muscular strain or emotion in persons over 50 years of age. Evidence of arterio-sclerosis, high-tension pulse, albuminuria, cardiac hypertrophy.
	EMBOLISM . . .	Sudden onset in a young adult or child, the subject of endocarditis.
	THROMBOSIS . . .	Gradual onset sometimes with prodromal symptoms of headache, etc., but often quite sudden: in male adults under 50, generally due to syphilitic endarteritis; in persons over 50, usually evidence of arterial degeneration and weak irregular heart.
	INFLAMMATORY POLIO-ENCEPHALITIS, SUPERIOR AND INFERIOR . .	Onset acute; headache, giddiness, vomiting, delirium, paralytic affection of eye-muscles or muscles supplied by neurones of bulb. History of chronic alcoholism or of some toxic condition.

of the recorded cases have ended fatally. Improvement, or even recovery, however, may take place. Rest and avoidance of all fatigue and excitement is most important; the patient should be kept in bed, and if there is any difficulty in deglutition or in mastication, minced food should be given. Great care should be exercised in using the tube for feeding. Strychnine, suprarenal extract, and thyroid tabloids have been tried without benefit; iron and arsenic have no specific action upon the disease, but may be employed as general tonics. Galvanism and massage also seem to be of no use, if they are not positively harmful.

CHRONIC OPHTHALMOPLEGIA.

Paralysis of the ocular muscles from peripheral causes has already been dealt with. The following description refers to a chronic progressive paralysis due to affection of the nuclei of the ocular muscles by a chronic inflammatory or degenerative process. Progressive ocular paralysis is sometimes associated with bulbar symptoms, showing that the degenerative process has affected the nuclei of other cranial motor nerves, and in a case of Hutchinson's, which Gowers examined, the microscopical appearances of the nuclei of the ocular nerves were very similar to the appearances presented by the anterior horns in progressive muscular atrophy. Gowers states, "thus the lesion in a fairly characteristic case was a degeneration limited to structures having a common function." Nuclear ophthalmoplegia in the pure form is seldom seen; it was first described by von Graefe, and later by Hutchinson, who also noted that in a number of cases (one-third) it is associated with optic atrophy.

The disease develops itself in the following manner: diplopia or ptosis occurs; gradually in the course of months or years, the paralysis spreads to other muscles of the eyes; very frequently the internal muscles of the eyeballs are spared, also the levator palpebræ is either incompletely paralysed, or sometimes escapes altogether. When the internal muscles of the eyeball are unaffected, nuclear paralysis can be diagnosed with confidence. Ophthalmoplegia exterior bilateralis can only be explained by a nuclear lesion.

It is not uncommon, however, for the internal muscles to be affected later on in the disease, owing to the extension of the process of degeneration to the superior nuclei; a group of cells smaller in size and situated higher than the other groups. In the fully developed disease, the eyeballs are immovable in the orbits, directed straight forward or somewhat outwards,

II. Chronic Bulbar Disease.	CHRONIC MENINGITIS .	Evidence of involvement of two or three nerve roots of bulb; previous history of syphilis.
	NEW GROWTHS AND ANEURYSM . . .	General symptoms due to increased intracranial pressure.
	DISSEMINATED SCLEROSIS	Absence of general symptoms of tumour. Pallor of discs. Coincidence of some characteristic brain and spinal symptoms with bulbar.
	PROGRESSIVE BULBAR PARALYSIS	Insidious onset; progressive character affecting first speech, then swallowing, etc., usually in the fifth or sixth decades of life. Symptoms of atrophic paralysis of muscles, sometimes combined with progressive muscular atrophy.
III. Myasthenia Gravis.	Absence of muscular atrophy, normal electrical excitability or the myasthenic reaction; considerable remissions; marked development of the paralysis by fatigue; bulbar symptoms most prominent, but all voluntary muscles more or less affected, especially extrinsic eye-muscles.	

and paralysed for all voluntary movements; in a few cases the upper nucleus of the facial has been involved, causing paralysis of the orbicularis palpebrarum.

Progressive ophthalmoplegia may constitute the whole of the symptoms of the disease, but more frequently it precedes or accompanies other widespread degenerative processes, such as locomotor ataxy and general paralysis, and in rare instances it may occur in bulbar paralysis, progressive muscular atrophy, combined sclerosis of the spinal cord, and multiple sclerosis.

A similar condition has been described as congenital and even hereditary in origin.

Prognosis is in most cases unfavourable. The disease may disappear spontaneously or under antisyphilitic treatment, but occasionally improves or remains stationary. It is too often the herald of some more serious general degenerative process.

Treatment.—In syphilitic subjects, mercury and iodide have seemed useful when the onset was acute or subacute; in cases that are chronic from the beginning, this treatment, as in locomotor ataxy and general paralysis, is useless. Nerve tonics which are suited to primary degenerative processes are indicated—strychnine and arsenic being the most effective. Galvanism, as in bulbar paralysis, appears to be of little use. In acute cases, leeches to the temples or blisters behind the ear may be applied with benefit.

F. W. MOTT.

AFFECTIONS OF SPEECH.

Under this head fall (*a*) Lisping; (*b*) slurring speech, as from alcohol and various other forms of nervous disease; (*c*) scanning speech, as in some cases of disseminated sclerosis; (*d*) stuttering. Of these, *b* and *c* will be found described under the various diseases in which they occur.

Lisping.

The imperfect pronunciation of various consonants. A certain amount of lisping is always present when a child begins to speak, and occasionally persists without any abnormality in the organs of speech, and the lisp becomes a personal or racial characteristic. *R* and *s* are the consonants most usually mispronounced, *w* or *sh* being substituted for them respectively. In some cases lisping is due to anatomical or functional defect in the closing of the palate, as in cases of cleft palate, diphtheritic paralysis, bulbar paralysis, etc. In these, owing to the failure of the soft palate to cut off the respiratory blast from the nasal passages, the voice acquires a nasal twang, and difficulty is experienced in the production of the "explosive" consonants, *p*, *b*, *t*, *d*, *k*, and *g*. *M* is substituted for *p* and *b*, and *n* for *t* and *d*. In rare cases there may be lisping on eight or ten of the consonant sounds, *t*, *d*, or *n* being substituted for them. As a result, the speech is quite unintelligible, although obviously divided up into words and sentences. It is only on carefully analysing the sounds that it can be found what letters are being mispronounced. This condition has been described as a separate affection, under the name *idioglossia*.

Treatment.—Patient, persistent practice is the only treatment required in most cases, except where there is a cleft palate or some other oral deformity. In cases of idioglossia, it is advisable to separate the child from those who have learned to understand his speech while he is under tuition.

STUTTERING.

A spasmodic affection of the muscles of articulation and respiration, leading to hesitation in uttering certain consonants. It occurs chiefly in childhood, and is twice as common among boys as girls. It is usually noticed when the child begins to talk, or at some epoch in his life, *e.g.* when he goes to school. Its causation is obscure. Direct inheritance is rare, although a neurotic family history is usually to be obtained. In a few cases the disorder is clearly to be traced to imitation of another stutterer.

Symptoms.—The phenomena vary in individual cases. The patient exhibits difficulty in pronouncing certain letters, when they occur at the commencement of a word or syllable. As he is speaking, he suddenly has difficulty in commencing to utter a word, and exhibits obvious effort. He either remains silent, or in some cases rapidly repeats the initial letter, speaking, for instance, of a b-b-b-ath, b-b-b-un. The word is suddenly ejaculated, and the succeeding words are very rapidly uttered, until the flow is checked by a fresh stutter, the rhythm being described as "like water being poured out of a bottle." This jerky utterance may persist after the stutter has been conquered. In the more severe cases, during the effort to speak, there may be associated sounds, or associated grimaces, or other movements which are extremely difficult to cure.

In most cases it will be found that there is a loss of the normal perfect co-ordination between the action of the expiratory muscles and the larnygeal and articulatory muscles, the laryngeal muscles being most at fault, so that the patient is straining his articulatory mechanism instead of vocalising. It is curious that this loss of co-ordination never occurs when the patient sings or intones.

The patient should be made to read aloud some unfamiliar prose, and the consonants on which he stutters noted. At the same time any errors of respiration, and any associated sounds or movements, will be detected. It is important to notice if there is any lisping on any consonant, as the removal of the lisp by constant practice will, in many cases, lead to the disappearance of the stutter.

Prognosis.—Many cases recover spontaneously, but recovery can be hastened by intelligent practice. A relapse often occurs when a boy goes into fresh surroundings, as when he goes to boarding-school, or goes to live abroad.

Treatment.—The nature of the complaint should be explained to the patient, who should be taught to familiarise himself with the proper position of the tongue, lips, etc., for the production of the difficult consonant, and should be made to understand that when he meets with difficulty he should direct his efforts to vocalising rather than articulation. Regular reading lessons, of at least half an hour twice a day, must be given and supervised by some responsible person. The reading should be, if possible, in a large room, so as to encourage the patient to raise his voice. During the lesson he should stand with the shoulders well thrown back, and keep the chest well filled. He must speak in a full resonant voice, as if trying to make his voice carry to a distance. On coming to a difficult consonant, he should be told to raise his voice and to direct his attention to pronouncing the following vowel, or if the consonant be voiced (like *b*, *v*, *g*, etc.), the vocal element of the consonant. A list should be made at each lesson of the words which give difficulty, and these must be diligently practised. Little progress can be expected for about a month, but after that progress is more rapid. Ultimate recovery is usually complete, but there may be recurrence in moments of excitement or anger.

W. S. COLMAN.

APHASIA.

The term aphasia is used to describe a complicated group of symptoms in which there is either—(*a*) difficulty in expressing propositions by speech (motor aphasia), or in writing (agraphia), not due to paralysis of the muscles concerned in articulation or writing respectively, but to disordered processes in the cerebrum; or (*b*) difficulty in understanding spoken or written words, without any defect in the sense of hearing or vision (sensory aphasia), the different forms being known as word-deafness or word-blindness. There may be perfect preservation of intelligence along with severe aphasia, although there is usually more or less impairment due to the lesion which has brought about the aphasia.

Anatomy and physiology.—The central processes concerned in voluntary speech are still imperfectly understood. Experimental observation on the lower animals is here impossible, and our knowledge of this important branch of physiology has to be obtained from observation of children learning to talk, and from clinical investigation of cases of aphasia in which there is a pathological condition of the centres concerned. But it is seldom that the lesions causing aphasia are so limited and so precise as to enable definite conclusions to be drawn from any one case; and, further, there is such a close functional connection between the different centres concerned, that extensive lesion of one always causes more or less disturbance of the others; and another difficulty lies in the fact that in the majority of cases there is at first considerable clouding of the mental faculties. Hence, there are very divergent views held by high authorities as to the nature of the central speech processes, and of the explanation of individual cases of aphasia. The diagrams which are employed only give a partial view of the subject, and it must be remembered that, except in their broad outlines, few of the views that are now held can be considered as finally proved.

The centres which are concerned in voluntary speech are usually described as four in number, and are situated, in right-handed persons, in the dominant left hemisphere. In left-handed persons, the centres are in the right hemisphere. The centres are as follows:—

Sensory	Auditory Word Centre (A)	Posterior third of the left superior temporo-sphenoidal convolution.
	Visual Word Centre (V)	Angular gyrus.
Motor	Motor Speech Centre (M)	Posterior third of the inferior frontal convolution (Broca's convolution).
	Graphic Centre (G)	Probably in the posterior third of the second frontal convolution and adjoining part of the ascending frontal convolution.

1. The auditory speech centre (A) is found in the neighbourhood of the auditory centre in the left temporo-sphenoidal lobe. Its exact extent is not known with certainty, but it occupies the posterior third of the left superior temporo-sphenoidal convolution in right-handed persons, and probably part of the supramarginal convolution. It is the first part of the cerebral mechanism to be educated in the child, and probably is the dominant centre in connection with speech processes throughout life. It is in close association with Broca's convolution by a definite commissure. Lesions of the centre or of this commissure produce an inability to understand spoken words, and, owing to the close connection between it and Broca's convolution, also entail more or less motor aphasia of the amnesic type.

2. The visual word centre (V) situated, in right-handed persons in the left hemisphere, in the neighbourhood of the auditory word centre, occupies the angular and part of the supramarginal convolutions. Lesions of this centre cause word-blindness, and also disturbance of the graphic word centre, so that the patient is no longer able to express himself in writing.

3. The motor speech centre (M) is in the posterior part of the left inferior frontal convolution. Its importance with reference to expression of words by speech was established by Broca in 1861, and it is usually known as Broca's convolution. In left-handed persons this centre is in the corresponding convolution on the right side. The inferior frontal convolution is larger and more complex on the left side. This centre is in close connection with the centres for the movements of the tongue and larynx, but is probably distinct from them. Its destruction gives rise to motor aphasia in its most complete form.

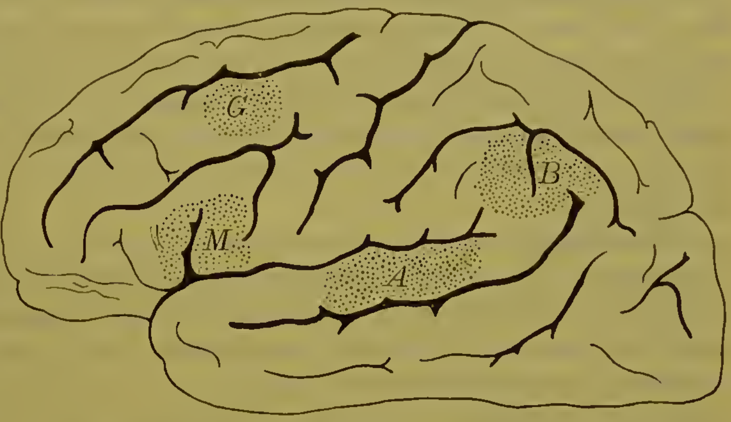

Fig. 24.—Diagram showing position of the various centres. *A*, Auditory word centre; *B*, visual word centre; *M*, motor speech centre; *G*, graphic centre (probable position).—After Bastian.

4. The motor graphic centre (G) is probably situated in the middle of the ascending frontal convolution. It is in close connection with the motor speech centre, and even if the arm is not paralysed, as it usually is from the accompanying right hemiplegia, there is almost always some agraphia if Broca's convolution is destroyed. It must be remembered that writing is a late acquisition of the human race, and, except in civilised countries in the present century, has been an accomplishment possessed by few, and hence this centre is less definite and independent than the others.

The connection between these centres and the higher intellectual centres is unknown. An independent naming centre has been supposed to exist by Broadbent and others, but this view is not now generally held.

Aphasia may therefore be produced by lesions of any of these centres, or of the strands of white fibre connecting them with one another, or by interruption of the motor path leading down from the motor centres in the cortex to the nuclei in the pons controlling the muscles of articulation.

Education of the speech centres.—In the new-born infant the use of all these centres is in abeyance. The first to become functionally active is the auditory word centre, the infant being able to understand words and even sentences for several months before there is the power to utter a single word. Later, he attempts to imitate the sounds which he has been taught to associate with certain objects, and the motor speech centre begins to take on its functions, but it takes months of practice before there is much facility in expressing ideas. It is probable that, in ordinary voluntary speech, the auditory image of the word is unconsciously evoked before the motor centre is brought into action. At any rate, the integrity of the auditory word centre is absolutely necessary for the correct expression of speech, for in all cases of lesion of this centre, or of the path from it to the motor centre, there is invariably more or less complete motor aphasia. When the child is about 3 or 4 years old, he is usually taught his letters, and to recognise words. He is generrally taught to say the word aloud, and in this way his visual word centre is educated through his motor centre; so that in motor aphasia it is almost always found that the reading of a newspaper becomes very difficult, the patient often having great difficulty in understanding what he reads, although he may comprehend if it is read aloud to him. Lastly, having learned to read, the child is taught to write, and his graphic centre comes into action. When learning to write, the child not only recalls the sensory images of the words stored up in his visual word centre, but also calls up the motor memories, so that he almost always frames his mouth for the production of the word, even if he does not actually say it aloud.

Etiology.—Since aphasia is usually met with as a complication of right hemiplegia, the most frequent causes are those of the latter disease, namely, hæmorrhage, thrombosis, embolism, abscess, gumma, or new growth, affecting the parts of the left hemisphere concerned in speech processes, or tumour or meningitis in the membranes over the centres. Aphasia is also seen as a transient symptom after some epileptic and epileptiform convulsions, and after attacks of *petit mal,* verbal amnesia being the most common form. It is also sometimes due to blood states (toxic aphasia). These may depend on the presence of substances formed in the blood, as in some patients suffering from typhoid fever, or the toxic agent may be introduced from without, such as lead, snake poison, cannabis indica, etc. In very rare cases aphasia is due to hysteria.

Symptoms.—**Motor aphasia.**—When this form is complete, there is complete inability to articulate a single word, although the patient makes meaningless articulate sounds, showing that the laryngeal and articulatory muscles are not paralysed, and he shows by his gestures that he understands what he wishes to convey, but is unable to express it in words. Often he can indicate by a pressure of the left hand the number of syllables in the word which he vainly tries to utter. Even if the word is suggested to him, although he vigorously gestures assent he is quite unable to repeat it. He can occasionally express himself in writing, but, as a rule, this is impossible, owing to accompanying right hemiplegia, or to involvement of the graphic centre in the lesion, which has produced the motor aphasia. Usually, though there may be no power of naming objects or of expressing a proposition, the patient may be able to say a few words or phrases in common use, such as "Yes," "No," "Oh dear," or "Bother," and when annoyed may be able to swear volubly. This power of speech is due in all probability to action of the corresponding convolution in the right hemisphere. Not infrequently the patient in answer to all questions uses the same phrase, which is spoken of as a recurring utterance. It is probable that this was the phrase which he uttered just before he was seized, as in the case of the librarian whose only words were, "List complete."

After a time, however, in the majority of cases, there is considerable improvement, and he begins to use a few words. Although quite unable to express himself in grammatical sentences, he has a number of familiar words and sentences at his command, and with the supplementary aid of facial gesture and pantomime he manages to convey his meaning to those around him. It will be found that he has more difficulty in giving the names of objects than with other words, and this is particularly noticeable when he is flurried or put out. In speaking he compresses his sentences, leaving out prepositions and conjunctions and superfluous words. Verbs appear to give rise to less difficulty than other parts of speech, but the grammatical inflection of the words is often erroneous.

Pure motor aphasia is best seen in limited lesions of Broca's convolution. When the lesion is larger, the commissures leading to the other centres are involved, and there is almost always a good deal of verbal amnesia Motor aphasia may also occur from interruption of the motor path to the articulatory muscles, as by a lesion of the internal capsule; but if the centre is not damaged, the aphasia is only transient, the impulses probably passing across the corpus callosum and descending by the opposite capsule.

Verbal amnesia is the term applied to a form of aphasia very similar to the foregoing. In this form, when complete, there is not only inability to express ideas in words, but there is inability to call up the word in the mind. It will be remembered that in cases of pure motor aphasia the patient was able to indicate the number of syllables in the word, but in verbal amnesia he is unable to do so. In incomplete cases there is forgetfulness of certain words only, especially names. When the patient is shown a pen, for instance, he may say, "That is a ——, oh dear, I can't remember," saying the words he can use without difficulty. Verbal amnesia and especially amnesia for nouns is seen in its most complete forms in lesions of the auditory word centre, but it also occurs in lesions of Broca's convolution, due to the intimate connection between that centre and the auditory word centre. Slight degrees are also seen in any cases where there is brain exhaustion, and as a temporary condition after a shock or a fall.

Paraphasia is the term applied to the condition in which a patient, in endeavouring to express himself, uses words which are not those which he intends to employ, and which are inappropriate. A slight degree is often present along with motor aphasia, and then the patient may recognise that the word is wrong and make distressed efforts to find the right one. But, as a rule, when it is at all marked it is an accompaniment of sensory aphasia, and due to a lesion of the auditory word centre or of the path from that to the motor centre in the frontal lobe. In this case the patient is usually unaware that he is using the wrong word. An extreme form of paraphasia is sometimes described as "gibberish aphasia," and is very painful to witness. The word-deaf person expresses himself in an unintelligible jargon, and has no idea that he is talking nonsense, and is distressed that what he says is not acted on by those around him. His speech is divided into words, and his intonation shows that he is arranging those words into sentences, but to a bystander it sounds much as though he were speaking fluently in some foreign language.

In slighter forms of paraphasia the patient uses actual words often arranged into sentences. These may have no connection with the idea which he wishes to convey, as, when a patient of Meynert's was asked, "How do you do?" replied, "My country is a beautiful one"; and when asked to put out her tongue, remarked, "My brother John." More often the word used bears some phonetic connection with the word intended, such as "water" for "watch," and "pink" for "pen."

Agraphia.—It is often impossible to trace how far inability to express words in writing may be due to affection of the special graphic centre, in those cases where there is paralysis of the right arm, as part of the hemiplegia, which has been caused by the same lesion as the aphasia. Owing to the late development of the faculty of writing, the centre is trained through all the other centres, and therefore there is always more or less agraphia, in forms of aphasia due to lesions of the other centres. It is most complete in cases of word-blindness. There may be every gradation from mere misspelling to complete inability to trace even his own name.

Word-deafness.—A person affected with word-deafness hears the voice of a speaker, but the word sounds no longer call up any meaning. In very complete cases he may not even recognise that he is being addressed in words, and pays no more attention than if his friend were coughing, or making any other inarticulate sound. Usually, however, he recognises that he is being spoken to, but the words convey no more sense to him than if the speaker were using a foreign language with which he is unfamiliar. Although there is this inability to understand what is spoken, the patients develop so much facility for interpreting facial expression or gesture, that they often appear to understand. In testing, therefore, whether an aphasic is word-deaf, it is important that the investigator should stand in such a position that his face cannot be seen by the patient. He should tell the patient to carry out some simple but unexpected command, such as "Lift up your left hand," avoiding such directions as the patient might expect, such as "Put out your tongue."

In slighter cases, or when the patient is recovering, he is able to understand much of what is said, although individual words, again chiefly the names of things, convey no meaning to him. The usual association of verbal amnesia and of paraphasia, with word-deafness, has been already referred to. Unless word-deafness is complete, there is usually power of repeating words which are spoken to him, sometimes in a parrot-like manner, sometimes with understanding of what is said. Word-deafness does not interfere with the copying of written words.

Word-blindness.—This condition, sometimes termed alexia, is due to loss of function of the visual word centre in the angular gyrus, or to interruption of the paths leading to that centre from the half-vision centres in the occipital lobes. A patient suffering from word-blindness is able to see the words and letters, but is totally unable to understand them. They look to him, in fact, much as a Russian book looks to one unfamiliar with that language. Where word-blindness is complete, there is in almost all cases agraphia. The exception is in cases where the angular gyrus is intact, and the aphasia is due to severing of the commissures between it and the occipital centres. There may be complete word-blindness without blindness for the individual letters, which are readily picked out; and even where there is complete blindness for words and letters, there may be ability to read figures and to add them up correctly. The patient should be tested for word-blindness by showing him a clearly-written direction, such as

"Shut your eyes." If he cannot understand this, he should be asked to pick out a *k* or a *y*, and his power of recognising numerals may then be tested. Care must be taken not to be misled through the patient picking out some word the shape of which has become familiar to him, such as his own name. Owing to the proximity of the auditory word centre, there is usually more or less word-deafness and verbal amnesia associated with word-blindness. Hemianopia is a rare complication, unless the lesion extends so deeply as to involve the optic radiations. The power of writing is always impaired, and usually lost. Owing to the word-blindness, the patient does not perceive the errors in what he has written.

Mind-blindness.—This symptom, including object-blindness, although not connected with speech, is so closely associated with word-blindness that it may be considered here. A man affected with mind-blindness is able to see objects and to avoid them, but their nature is not perceived. A man in this condition may be unable to recognise the faces of any of his friends, although he at once recognises them when they speak. Even if he is shown his clothes, he looks helplessly at them, but has no idea of their use till he touches them. A dog in whom the condition has been experimentally produced, although able to run about and avoid obstacles, does not recognise even his food until he is made to smell it.

Mind-blindness can be produced experimentally, by removal of both angular gyri in dogs; and in several recorded cases in man, where there has been an examination of the brain, a lesion has been found on both sides of the brain, in the supramarginal and angular gyri.

Localisation.—It may be well now to consider the different forms of aphasia which may result from lesions in different parts of the brain. In the first place, it must be remembered that lesions, especially sudden lesions, in any part of the left cerebral hemisphere, may be accompanied by transient aphasia; secondly, an extensive lesion, especially if suddenly produced, of any of the centres concerned in speech, will cause extensive disturbance of the other centres, at any rate during the first few days. Thus, as has been mentioned, in any large frontal lesion, causing motor aphasia, there is also some disability of the visual word centre, so that the patient is no longer able to read his newspaper.

1. *Lesions of the third frontal convolution* (*motor speech centre*).—If the lesion be small, there may be pure motor aphasia; but this is rare, and usually there is much verbal amnesia. Writing is nearly always interfered with, owing to involvement of the neighbouring graphic motor centre, or to interruption of the commissural path btweeen the two centres. A subcortical lesion, if immediately below the grey matter, causes exactly the same effects as a lesion of Broca's convolution itself.

2. *Lesions of the second frontal convolution* (*motor graphic centre*).—If these are small, pure agraphia is produced; but this is very rare. When the lesion is of any size, there is always paralysis of the arm, so that the amount of agraphia cannot be tested. Lesions usually encroach on the motor speech centre, so that there is motor aphasia as well.

3. *Lesions of the superior temporo-sphenoidal convolution* (*auditory word centre*).—In the case of small lesions, not completely destroying the centre, there is temporary word-deafness, which, as a rule, passes off in a short time; the most prominent symptom in partial lesions of the auditory word centre being verbal amnesia and paraphasia. In more extensive lesions there is not only word-deafness, but so much amnesia that the patient is almost speechless. Owing to the proximity of the visual word centre,

there is usually a good deal of word-blindness, and consequently of agraphia.

4. *Lesions of the angular region (visual word centre).*—These give rise to word-blindness, and consequent agraphia. If there be a lesion in the opposite angular gyrus as well, there may be mind-blindness in addition.

5. *Lesions in the neighbourhood of the island of Reil.*—Lesions in this position are apt to interrupt the commissure between the auditory word centre and the motor speech centre in the frontal lobe. In that case there is usually marked verbal amnesia, but paraphasia is the prominent symptom.

6. *Lesions involving the pyramidal tract.*—These rarely cause more than temporary aphasia. A way is soon established between the motor speech centre and the articulatory centres in the bulb, through the corpus callosum and the internal capsule of the opposite side.

Prognosis depends chiefly on the amount of destruction of the centre. If the centre be completely destroyed, there is little chance of recovery of speech, unless the patient be a child, when there may be some hope of educating the corresponding centre in the right hemisphere. When the lesion is only partial, great improvement, or even complete recovery, may take place. Where aphasia is due to pressure from a gumma of the membranes, complete restoration of function may be expected if anti-syphilitic remedies are given early. Capacity for re-education is greater in the case of the sensory centres than in that of the motor centres.

Treatment.—Treatment of the lesions causing aphasia being the same as for those causing hemiplegia, are referred to in that connection. As regards the speech defect itself, the only plan is to re-educate the patient, exactly as a child is taught to speak, read, and write. The process is a very slow one, and requires intelligent supervision and unwearying patience.

W. S. COLMAN.

DISEASES OF JOINTS AND MUSCLES.

ARTHRITIC MUSCULAR ATROPHY.

Etiology and pathology.—Few facts have been ascertained regarding the condition. In one case, the only change was in the muscles, and consisted in a narrowing of the fibres: a few presented longitudinal striation. Indications of inflammation have been found in the joint nerves, but visible changes in the spinal cord have never been discovered. We must also recognise the possibility of simultaneous neuritis, with acute changes in the nerve fibres; but this is exceptional. For ordinary arthritic atrophies, however, some other explanation must be sought. Paget long ago suggested a reflex cause, and this theory receives important support from the fact that the wasting of the muscles is prevented by previous division of the posterior spinal roots. The increased knee-jerk, etc., and the fact that arthritis may set up a degeneration of the spinal cord, are significant, and suggest that the influence from the joint nerves acts on the controlling structure of the muscle reflex centre.

Symptoms.—Articular inflammation is almost invariably attended by rapid wasting of the muscles that move the joint. Those that waste are chiefly the muscles which extend the affected joint. Thus, if the knee is inflamed, the muscles in front of the thigh waste; if the ankle is affected, the calf muscles waste; the deltoid, when the shoulder joint is inflamed. In rheumatoid arthritis of the finger joints, the wasting is usually conspicuous in the interossei. If the onset of the joint affection is acute, wasting occurs rapidly. It affects the whole length of the muscle; but if the muscle is wide and a compound one, some parts may suffer more than others. When the joint has recovered, the muscles, in most cases, slowly regain their normal size. The electric irritability of the atrophied muscles may be normal, or it may be slightly lowered, but the change is trifling. There is generally a distinct local increase in myotatic irritability. Occasionally this extends beyond the region of atrophy, so that a foot-clonus may be obtained when the knee joint only is affected. Very rarely some contracture occurs in the opponents of the atrophied muscles. Sensory symptoms are absent as a rule, but there may be slight tingling in the skin, and it is said that areas of anæsthesia sometimes develop. In such cases inflammation has probably extended to nerves in the vicinity of the joint.

Diagnosis and treatment.—A moderate degree of wasting, with evidences of joint affection, are usually sufficient to distinguish this condition. When the inflammation is brief in duration, recovery may be anticipated. Even if slight wasting is persistent, normal power is usually recovered, though the symptoms may continue for years. Treatment is local—electrical stimulation to the muscles and gentle rubbing. The muscles react readily to faradism, and a mild faradic current is probably the best form of stimulus to employ. All painful stimulation of the sensory nerves should be avoided.

MUSCULAR ATROPHY FROM OVERUSE.

The occurrence of such a condition need only be mentioned. The affection is seen most frequently in the small muscles of the hand, especially in persons of weakly constitution who use those muscles unduly.

We do not know whether this wasting is purely local, or whether the related ganglion cells fail first. The wasting often persists when the overuse is discontinued. Hypertrophy from overuse does not usually give place to atrophy. These two facts are somewhat in favour of the view that the lesion is primarily of the nerve cells. Wasting in these cases shows no tendency to extension beyond its usual limits, and the treatment consists in a cessation of the excessive exertion of the muscle, the administration of nerve tonics, especially strychnine, and the gentle electrical stimulation of the muscles by either faradism or voltaism.

MUSCULAR DYSTROPHY.

The so-called pseudo-hypertrophic variety of muscular affections has been recognised under this name ever since its graphic description by Duchenne. The features were muscular weakness and associated enlargement of sometimes few, sometimes many muscles. Duchenne also described another form of paralysis which he supposed to be similar in its etiology to the progressive muscular atrophy of adults, the *atrophie musculaire progressive* *de l'enfance.* This was characterised by wasting of the muscles of the face as well as of the limbs, and also by its tendency to run in a family. Leyden's attempt to separate the hereditary form of muscular atrophy into a distinct variety, need only be referred to. The next most noteworthy addition to our knowledge of these cases was the lecture by Gowers on pseudo-hypertrophic paralysis in 1879. In this a full description of the disease was given, with numerous clinical observations, and all that was known regarding its pathological anatomy was clearly set out. Gowers clearly recognised that hypertrophy, or seeming hypertrophy, and atrophy may be combined in different proportions, and that there are cases which conneet the two extreme conditions. In this way we get, as it were, a glimpse of the group which Erb afterwards clearly outlined, under the name of the juvenile form of progressive muscular atrophy. This was characterised by its occurrence in young people under 20, and by the fact that there was enlargement of some muscles, with wasting, especially of those about the shoulder. From a consideration of the characters and histories of those cases, Erb concluded that the three varieties, his own juvenile form, the hereditary form of Leyden, and pseudo-hypertrophic paralysis, were but examples of one condition, for which he proposed the name, *dystrophia muscularis progressiva.* Soon after this, Landouzy and Déjerine published an exhaustive paper on what they called *myopathic atrophique.* It dealt with observations of an exceedingly interesting group, the essential characteristics of which were widespread muscular atrophy commencing in the face, without hypertrophy. These cases were really identical with those described by Duchenne as the progressive muscular atrophy of childhood, but the later observers completed the description by the discovery, by post-mortem examination, that the disease did not depend upon, nor was it associated with, changes in the spinal cord, but that it was a muscular affection, without detectable nervous lesions, and therefore different from progressive muscular atrophy. They were led to regard their cases as a distinct and separate variety of myopathy, relying upon the commencement in the face and the absence of hypertrophy, and they refused to recognise the essential identity of their cases with those of Erb, in spite of the fact that in one of their quoted cases the face was not affected, and considerable enlargement of the calves, if it was not present at the time, had been noted a few years before.

The latest and most important contribution to the literature of this disease is again the work of Erb. In this he submits to examination numerous clinical records of cases of the different kinds, and makes it clear by the observation of cases of different types among members of the same family, by the description of cases which form distinct connecting links between the different varieties, and finally by the observation of the histological conditions in the affected muscles, that all the four varieties to which we have referred, namely, pseudo-hypertrophic paralysis, the hereditary form of Leyden, the juvenile form of Erb, and the Landouzy-Déjerine or facio-scapulo-humeral type—*atrophic musculaire de l'enfance* of Duchenne—are essentially the same disease.

Etiology.—Our knowledge of the causes of this disease is limited to a few general facts. Males furnish the majority of cases, and when females are affected the malady is said to be slighter in degree and later in development. There may be a tendency in a family to the affection of one sex and not the other; but, on the other hand, children of both sexes may suffer in the same family. In many instances, even in which several members of one generation were affected, no antecedent cases could be traced in the family. In other families antecedent cases can be traced, and these are invariably on the mother's side. The disease is thus transmitted by women who are not themselves its subjects. Indirect hereditary tendencies, such as are indicated by diseases of the nervous system, cannot be traced with sufficient frequency to give them any significance. The consanguinity of parents seems to become effective, only when raised in energy by repetition. The disease usually manifests itself during the period of development, but occasionally cases arise about the age of 20, in patients who have hitherto been not only active but actually athletic. In one case the patient, up to the age of 18, had been extremely active, and had won numerous prizes as an athlete, but before he was 24 he was completely helpless.

Morbid anatomy.—It is rare at the time of death for any muscles to be actually larger than natural. They are pale yellowish in colour, often to the naked eye resembling masses of adipose tissue. As seen under the microscope, this appearance is confirmed, for nothing may at first be visible except fat cells. On closer examination fibrous tissue is revealed, and also muscular fibres. The fibres for the most part preserve their transverse striation, although occasionally granular degeneration has taken its place. Very rarely in some part of a muscle there has been only wasting of the fibres, without the interstitial change present elsewhere. In some instances the interstitial tissue may be almost entirely fibrous, a few fat cells here and there being the only ones visible. Muscular fibres larger than normal are seldom found after death, but such fibres have been seen in fragments removed during life. It is possible that the increased size was due to a vital contraction, under the mechanical stimulus of the harpoon trocar with which the removal was effected. The motor nerves when examined have been found normal, and the spinal cord has been unaltered in most cases in which it was examined. In some, there have been slight and irregular degenerative changes. Hæmorrhages have occasionally been found, and the neuroglial cells have been increased in number. The fibres of the white substance have been found irregularly changed, but not so uniformly or so constantly as to give the changes any significance.

Pathology.—The significance of the pathological anatomy is, that the malady is a primary disease of the muscles. The disease is congenital, the result of a perverted tendency of growth inherent in the embryo, and derived from the germ from which the embryo proceeds. The difficult question of the precise mechanism by which the muscular fibres suffer must be regarded as an open one. In some cases of the pseudo-hypertrophic variety there seems to be a tendency to the formation of fat in the early stages of the disease, and its removal at the later stage. In others, the atrophic variety, the tendency seems to be towards simple atrophy; while, in other cases, we have the two processes combined. The latissimus dorsi and the lower half of the pectoralis are said to be sometimes congenitally absent in this disease. The loss of the knee-jerk is sufficiently explained by the lesion of the muscles.

Symptoms.—Impairment of power usually attracts attention before any peculiarity is noted in the appearance of the muscles. This impairment is first manifested in the difficulty which the subject of the disease has in going upstairs. At first, progress is slow, but of the usual character; later, the patient gets one foot on the step by laboriously hanging on to the banisters and dragging the other up beside it. But, after a time, it is quite impossible for the patient to ascend. This inability may be quite marked, while walking on the flat or going downstairs is still quite easy. But, as the patient walks, it is noted that his back gradually becomes more arched, his shoulders are thrown back, and there is a tendency to walk on the toes. At this stage, if he is placed on the floor, there is a very great difficulty in rising to the erect posture, and certain characteristic attitudes are assumed in the course of this attempt. It may be that the patient is unable to get off the floor without help. He has usually not sufficient power to extend the knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power acts at least advantage. He therefore places his hands on his knees, and his arms thus bring much of the weight of the upper part of the trunk on the femur, close to the fulcrum, between this and the power, which can then act at greater advantage. Moreover, the mere weight of the head, which is in front of the arms, tends to aid the extension. When the knees are extended, the power of the extensors of the hip may be sufficient to raise the body into the upright position; or the patient may aid them, usually it is so, by an upward push, as he takes the hand off the thigh. If, however, these extensors are weak, the hands are often moved higher and higher up the thighs, grasping alternately and thus pushing up the trunk. To get the requisite support, the knees must not be quite extended, and if their extensors have no power, the device cannot be employed, and the patient is altogether unable to rise. As a rule, the arms are not affected in the same degree as the legs. Often the forearm and the hand remain strong and normal when the upper arm is wasted, and the muscles about the shoulder especially, extremely atrophied. This weakness of muscles gradually, it may be very slowly, increases, and finally, in the majority of cases, the patient becomes quite helpless. The diseased muscles are weak, but the impairment of power is to some extent irrespective of the change in size. Those that are abnormally large are generally stronger than those that are much wasted, and in the latter the weakness increases with the wasting. In the legs the greatest weakness is in the flexors of the hips, the extensors of the knee, and the extensors of the hip. The muscles below the knee usually retain considerable power for a long time. The extensors of the ankle fail before the flexors. In the upper limbs, the depressors of the arms are usually alone weak in the early period of the disease, but subsequently the shoulder muscles suffer, then the triceps and biceps, while those that move the hand commonly retain good power to the last. The shortening and contraction of certain muscles lead to another group of symptoms—distortions, the result of permanent alteration in the position of joints. Some of these are produced by shortening of the less affected opponents of the weaker muscles. Thus the knee-joints become fixed by the contraction of the flexors, and the elbow by contraction of the biceps when the triceps has lost all power. These contractures, especially in the lower limbs, often lead to an earlier loss of power of walking than the disease itself really necessitates, and in rare cases tenotomy has been able to restore for a time the ability to walk. Another deformity, which is the result chiefly of muscular weakness, is curvature of the spine: an antero-posterior curve with the concavity backwards is an early symptom of the disease, and it may be extreme, so that a line from the scapula falls an inch or more behind the sacrum. This is really the result, not so much of the weakness of the trunk muscles as of the extensors of the hip, in consequence of which the pelvis is inclined forward, carrying with it the lower lumbar vertebræ. Hence, when the patient sits, the pelvis is supported on the ischial tuberosities, and the lordosis disappears. Electric irritability of the muscles is only altered when distinct weakness or wasting has set in, when it is lowered alike to faradism and voltaism. The knee-jerk may be at first normal, but becomes lessened and gradually disappears. Sensation is unaffected. The sphincters act normally in the vast majority of cases. All other functions of the nervous system are commonly normal, and the mental development of the subjects of this disease is not, in the majority of cases, materially different from that of other children of the same age. Occasionally, however, there is a congenital state of mental weakness, due apparently to defective development of the brain.

The progress of the disease varies very much in different cases. When it begins in early childhood, before the age of 7 or 8, the child is usually reduced to a condition of extreme helplessness. But this is not always the case, for one case is known to the writer of a man now over 40, who has never been able to run, who exhibits a typical picture of the pseudo-hypertrophic variety of the disease, and whose condition has certainly not become worse in the last ten years. Even if the disease commences later in life, the rapidity of the progress does not seem to be materially modified. It is usually fairly rapid, and ultimately ends, at all events in by far the largest number of cases, in more or less complete helplessness. It seems to be almost the rule that the cases in which the face is affected commence in later life, that is to say, that young adults are the patients who are most frequently affected. In this particular variety, also, the progress seems to be not quite so rapid as in the more common variety. It should be mentioned here that the affection of the face is usually shown by weakness of the orbicularis palpebrarum and in the orbicularis oris, so that the patient is unable to close the eyes completely or use the orbicularis oris in such a way as to produce an audible whistle.

Diagnosis and prognosis.—The diagnosis is usually easy. The peculiarity of gait, the mode of rising, the wasting, or the pseudo-hypertrophy, are all points which it is not easy to miss. The mode of rising is not absolutely pathognomonic, it has been noted to occur in commencing double hip-joint disease. Of the condition of muscles, that which is most characteristic is the combination of enlargement of the infraspinatus with the wasting of the latissimus and lower part of the pectoralis. The disease with which confusion is most common is the so-called congenital spastic paraplegia; both diseases affect children, in both there is weakness of the legs, but in the latter there is rigidity with exaggeration of the reflexes, and none of the typical localised wasting or pseudo-hypertrophy. In the case of any child with this disease the prognosis is naturally most grave. It is certain, or almost certain, that each year will bring increasing disability, and that the patient will not reach adult life, yet, as has already been mentioned, there are occasional exceptions to what is undoubtedly the rule. When the disease develops late, and the symptoms do not become considerable until after 20 years of age, there is a possibility that it may not attain its extreme degree. Even in such cases, however, this hope is seldom realised.

Treatment.—Unfortunately, so far, no mode of treatment which has been tried seems to have had any effect at all in arresting this disease. Cases in which careful gymnastic, electrical, and other forms of mechanical treatment have been carried out, have, in spite of it all, become steadily worse. We have already referred to the surgical procedures, in the shape of tenotomy, which are occasionally effective in giving a patient a few months longer of the ability to get about. Treatment by the administration of thyroid and thymus have both been tried, but, so far as we are aware, without any good result. Undoubtedly it is of importance to maintain locomotion as long as possible, and a moderate degree of muscular exercise, active if possible; but if this is impossible, then passive exercise is very desirable in every case of the disease. On the whole, however, it is a condition in which treatment is by no means hopeful.

PERONEAL TYPE OF FAMILY ATROPHY.

This is a disease, or rather a variety of disease, which is still somewhat indefinite, in both its pathological character and its symptoms. The cases described present certain resemblances to the idiopathic atrophies just described, yet the distribution of the wasting involves a constant difference from those affections, and some of the cases present features that indicate neuritis. The condition may have as an antecedent an acute specific disease, males suffer about as frequently as females. It generally begins in the second half of childhood, and very seldom after 20. It is occasionally hereditary, still more frequently collaterals suffer; the wasting is first apparent in the extensor longus hallucis or extensor communis digitorum, or in the peronei muscles. It probably occurs even earlier in the small muscles of the foot, where it readily escapes observation. The calf muscles suffer subsequently, and still later those of the thigh. The arms in most cases are only invaded some years after the legs, the intrinsic muscles of the hands being first attacked, and subsequently those of the forearm. The atrophy is often symmetrical, and the claw-like deformity of the fingers may result from the affection of the intrinsic muscles of the hand. The affected muscles occasionally present slight fibrillation. Electric irritability is diminished early, and faradic reaction may soon become extinct. Pains occur in some cases, the muscle reflex action is lost in the affected regions, and the cutaneous reflex action presents considerable variations. Vasomotor disturbance is said sometimes to occur. The nature of those cases is still doubtful. Peripheral neuritis, of extremely chronic course and peculiar origin, must be recognised as the most likely lesion, and it has been actually found in some cases by Friedreich, Gombault, Dubreuilh, and others. It is asserted by Hoffman that not only are the peripheral nerve fibres the seat of a primary degeneration, but that this change ascends the nerves at the anterior and posterior roots, involving the elements of the spinal cord.

It should also be remembered that the atrophy may be idiopathic in some cases. If the lesion is really neuritic, we can understand that an acute specific disease like measles should occasionally be its excitant, though its occurrence in families shows that a congenital tendency must be called into activity by the virus. It is especially important to note the occurrence of cases that occupy an intermediate position, and combine the characters of this disease and those of the myopathic types. Such combined cases are usually partial only, but such partial cases when combined ultimately cover the whole intermediate ground. For instance, the condition of the legs in the peroneal type has co-existed with that of the arms in the juvenile form in several recorded cases. The progressive character of the cases is commonly pronounced, but even if slow, it renders the prognosis gloomy, and the scope for treatment small, So far as anything can be done, it is by the measures already mentioned in the account of the treatment of the maladies to which the cases approximate, namely, primary neuritis on the one hand, and idiopathic muscular atrophy on the other.

FAMILY FORM OF MUSCULAR ATROPHY, IN CHILDREN WITH SPINAL LESION.

The above title best describes a series of cases described in the last ten years by Werdnig and Hoffmann in Germany, and Alexander Bruce and John Thomson in this country. The condition is characterised by the gradual onset, usually about the age of 7 to 10 months, in children previously healthy, of weakness in the hips and back, which gradually extends, so as to affect the arms and legs, and even the hands. Muscular wasting, with changed electrical conditions of the muscles, but without fibrillary tremor, is very marked, but no hypertrophy has yet been noted. The deep reflexes are lost, occasionally also the superficial ones, and sometimes symptoms indicating the implication of bulbar structures, have been superadded. No definite sensory impairment has been observed, although in one case there was a peculiar and marked insensitiveness to the faradic current. As a rule, more than one member of a family is affected, but isolated cases also occur. Death usually takes place from exhaustion, but often it is deferred until as late as the sixth year.

The symptoms enumerated show the relations on the one hand to progressive muscular atrophy, and on the other to the myopathies. Pathologically, the relations become more complex, for whereas wasting of anterior nerve cells and anterior nerve roots is distinct, and simple atrophy of muscles also, there are occasionally present hypertrophied muscular fibres, similar to those in pseudo-hypertrophic paralysis. The occurrence as a family disease is also significant in reference to its relations to the myopathies. And future observations may show that it is a type connecting myelopathy with myopathy, and consequently partaking of the characteristic features of each.

Treatment unfortunately is not very efficacious, and considering that the condition is probably one in which there is an inherited tendency to early death in certain closely related structures, this is not to be wondered at. Strychnine, by the mouth or hypodermically, galvanism, massage, and passive exercises are the therapeutic measures which suggest themselves. Nutritious food and good hygienic surroundings are of course essential.

MYOTONIA CONGENITA.

Syn., Thomsen's Disease.

This disease, described by the observer whose name it bears, and who is himself a sufferer from it, is characterised by a peculiar rigidity of the muscles, which comes on when they are first put in action after a period of rest. The rigidity is transient, and when it has once passed off, does not return so long as the exertion is continued.

Etiology.—It is often hereditary, usually affects several members of the same family, and both sexes suffer. It appears to be more common in Scandinavia and Germany than in France or England, but it is a rare disease.

Pathology.—No examination of the nervous system has yet been made, but the evidence of hypertrophy in the affected muscles, specimens being obtained by the excision of fragments, appears to be conclusive. It is true that this method involves a fallacy, that the stimulus of excision may possibly account for some alteration in the state of the muscle, yet in the cases examined by Erb, where the minimum width was the same as in health, the maximum diameter was double the normal, a difference which seems to preclude the possibility of its not being a real one. Most writers on the subject have followed Leyden in regarding the malady as essentially muscular in nature, and consisting in an altered functional condition of the muscular tissue. But we must remember that the malady has apparently resulted, in rare cases, from influences acting on the nervous system in adult life, and also that in congenital cases the spasm only develops, as a rule, after some years of voluntary action. It may thus be an acquired disease, and similar spasm at least may be due to a primary affection of the cord. The phenomena of Thomsen's disease really suggest that the cells respond abnormally to the voluntary stimulus, that this causes at first increased tonic activity, slowly ceasing as their energy is lessened by action. We must remember that the muscular and nervous tissue have some conditions in common, and it is conceivable that an abnormal functional state, congenital in origin, may be common to the two, and that the peculiar over-action in the muscles may be accompanied by a similar over-action in the ganglion cells of the spinal cord, or even in the pyramidal cells of the cerebral cortex. It is even conceivable that the condition of the nerve cells may be a primary change, and that of the muscles secondary; although when produced it is in some degree independent, and may be excited independently by local stimulation.

Symptoms.—In most cases the symptoms have been first noted in childhood, and they increase during the period of muscular development. In some cases, after some exciting cause, symptoms apparently identical with those of the congenital disease have come on in early manhood. Severe and prolonged exertion preceded the onset of very characteristic symptoms in one case without heredity. The characteristic symptom is the tonic spasm in the muscles, which occurs, as we have already stated, when they are put into action after a period of rest. As soon as the patient attempts to move, the muscles become rigid, but after a few minutes, or less, the spasm passes away, to be renewed in slighter degree by a fresh attempt. If the attempts are continued, the spasm soon becomes trifling, and does not return again until after a period of rest. It is worse in cold and in damp weather, and is sometimes lessened by alcohol, and is increased by attention and by fear of it. The arms are usually less affected than the legs, and the muscles of the face are generally free. Mastication may, however, be interfered with by spasm in the muscles of the jaw. Rarely the tongue or face has been affected, still more rarely the muscles of the eyeball. Most voluntary muscles, indeed, are liable to suffer, even the muscles of the pharynx and the larynx, and those concerned in the processes of respiration, defæcation, micturition, and coitus have also been involved. The muscles are always well nourished; often, indeed, above normal size, and sometimes possessing more than normal strength. They are, however, sometimes weak. Mental irritability and hypochondriacal tendency are not uncommonly conspicuous in some cases. Momentary electrical stimulation of the nerves causes only a momentary contraction of the muscles, but continued stimulation always causes a prolonged contraction, the myotonic reaction of Erb. In certain muscles, however, an interrupted current caused peculiar wave-like contractions, about one per second, passing from the negative to the positive pole. Any strong stimulation of the muscle itself caused prolonged after-contraction. The irritability of the nerves is generally normal in degree, that of the muscles normal or increased to voltaism. The sensibility has been intact in all undoubted cases of the disease, and the superficial reflexes have been unaltered. Myotatic irritability has been normal or increased. Once developed, the disease seems to persist with little change throughout life, but in one case, a female, in whose family there were other cases, considerable improvement is said to have followed marriage. In some instances the symptoms are very slight, and remain so even when they begin in early life, and occur in the same families in which severe cases are met with.

Treatment.—No treatment, properly so called, appears to exert any influence upon the disease. The congenital malady persists through life, although, in certain cases, as we have said, it seems to have been ameliorated by a life of active muscular exertion.

CONGENITAL PARAMYOTONE.

This is a strange family affection, allied to Thomsen's disease, which was widely spread in the affected family, and could be traced through six generations. In some individuals it was manifested immediately after birth by tonic spasm, lasting from a quarter of an hour to several hours, excited chiefly by cold. The rigidity was followed by temporary weakness. The face muscles were very prone to become thus rigid, especially the orbicularis palpebrarum and oris. In the legs the rigidity was slighter than in the arms, but the subsequent weakness equally marked. Warmth removed the spasm. Eulenburg, who described this strange family affection about ten years ago, speculates that the symptoms may be due to reflex vasomotor spasm in the muscles, because the diminution of blood supply to muscles renders them weak.

ATAXIC PARAMYOTONE.

In the case to which this designation has been applied there was persistent tonic spasm, like the transient spasm of Thomsen's disease, associated with distinct ataxy and also weakness and anæsthesia. The symptoms commenced gradually in the legs, in a healthy man, æt. 40. It invaded the arms six months later, it was present on waking in the morning, and through the day interfered with all movements. Prolonged exertion had no influence on the spasm, which resisted passive movement as well as active. Power in the arms was diminished, the extensors of the fingers were weaker than the flexors, but the electric irritability of the muscles seemed to be normal. Inco-ordination was especially marked in the hands, and no trace of the knee-jerk, or other evidence of myotatic irritability could be elicited. The spasm, however, was sufficient to account for this, so that its real condition is uncertain. The mind, special senses, and cranial nerves were unaffected.

MYOCLONUS MULTIPLEX.

There is little doubt that under this name, since it was some years ago applied by Friedreich to a condition in which spasm was present in the muscles on both sides of the trunk, and also the extremities, many affections have been described which are really examples of different forms of choreic movements, chiefly functional in character. In the true disease the muscles most involved are those of the trunk and the upper limbs. The spasms, which occur suddenly, are usually bilateral, not necessarily symmetrical, and may be so powerful as to throw the patient down. They occur at intervals, sometimes of only a few minutes, but sometimes an entire week will elapse between paroxysms. The movements vary from a few to as many as 180 a minute. There is no mental disorder, the tendon and skin reflexes are increased, and speech may be interfered with.

The prognosis is usually favourable, although in some instances the disease has proved fatal through exhaustion.

In regard to treatment, attention to the general health is of the chief importance. Galvanism and static electricity are said to have been found useful, but it is to be doubted whether they are as efficacious as they have been supposed to be.

NEURO-MYOSITIS.

Under the name neuro-myositis an affection has been described, usually confined to a single limb, generally the arm, in which symptoms indicating an interstitial rheumatic inflammation of the muscles are the leading feature. They become tender, but the especial character is the pain caused by contraction of the fibres. The adjacent fibrous tissue, especially that about the joints, may also suffer, and the condition may closely simulate that of brachial neuritis. It occurs chiefly in patients, in the second half of life, but is also met with in young persons, even children, as a sequel to rheumatic fever. It may be limited to the structures and muscles of the hand, but it more frequently commences in the neighbourhood of the shoulder, passing down the arm. The change in the intrinsic muscles of the hand, and in the tissues about the tendons, may last so long that the joints become fixed before there is time for the restoration of the muscles. But some œdema in the acute stage may be followed by peeling of the epidermis, and wasting of the subcutaneous structures. The chief feature of the condition is the affection of muscles, shown by the production of pain in them by those forms of stimulation, namely, compression by voluntary contraction, and passive tension in the antagonist, to which nerve-endings normally respond. There is usually some tenderness on sudden compression of the muscular substance, but often this sensibility is inconspicuous. The arthritic changes, which may be severe within the joints, produce adhesions which, with the alterations in the capsule, fix the joint in an abnormal posture. The malady is commonly of prolonged duration, developing insidiously from an initial acute attack of local rheumatism, and the resulting interference with the use of the part is greater than is proportionate to the amount of rheumatic inflammation. After recovery, some chronic neuralgic pain is apt to remain in the affected limb. Great care is necessary, especially in regard to local therapeutic measures, so that nothing may be done which is likely to increase the pain, as the movements necessary may then become quite impossible. Gentle upward rubbing to the muscles should be combined with slow passive movement, at first scarcely appreciable in degree, and continued with the endeavour to keep behind the production of the muscular spasm. This will often enable us to effect much relief by local applications of dry or moist heats, anodyne liniments, etc. The internal treatment is such as is suitable for the gouty and rheumatic constitutional state.

POLYMYOSITIS.

In rare cases there occurs an acute interstitial inflammation of the muscles, similar in many of its features to ordinary multiple neuritis, differing, however, in the fact that the affection of the nerve fibres is not only slight and subordinate, the muscles themselves being the seat of the chief change. The condition is symmetrical, a condition indicating the blood as the vehicle of the exciting toxic substance. The subcutaneous fibrous tissue is usually also inflamed, causing œdema and dermatitis, and although, as we have said, in the majority of cases the nerve fibres are slightly and subordinately affected, in some this affection of the nerves is much more obvious, and the distinction from polyneuritis can scarcely be made. The malady is so uncommon, and the cases which have been met with have varied so much, both in severity and in precise causation, that it seems unnecessary to do more than merely call attention to the fact that such a symmetrical disease of muscles has been met with under conditions suggesting toxic origin, similar to that which produces closely related disease of the nerves.

JAMES TAYLOR.

FUNCTIONAL NERVOUS AFFECTIONS.

NEURALGIA.

By this term is meant pain in a nerve, a symptom not necessarily associated with any obvious pathological change. Many cases of neuralgia, on the other hand, would appear to be due to an inflammatory condition of the nerve sheath—perineuritis; as illustrations of this may be mentioned sciatica and brachial neuralgia or brachialgia.

Neuralgia should at the outset be distinguished from neuritis; the former signifying nerve pain, the latter inflammation of nerve fibres. The characteristic features of neuritis lie in trophic disturbances of the skin over the distribution of the inflamed nerve or nerves, and atrophy of the muscles with characteristic alteration in their electrical excitability. Pain is a symptom common to both affections: in neuralgia it is the only symptom; and hence it comes to be regarded as "the disease."

Both troubles are associated with the presence of tender points in the position of the nerves and their branches. In the neuralgic form, the pain is usually limited to certain spots, where the cutaneous branches penetrate bony canals or fibrous rings (points douleureux de Valleix); in the neuritic form the pain is more marked along the trunks of the nerves.

Symptoms.—Neuralgia is met clinically in two forms: either as a local condition, in which pain is limited to one or more nerves, to which the attention of the physician is directed; or, secondly, as an element in a state of general nervous debility, where most of the nerves reaching the surface present painful points on pressure. This latter variety is common and often overlooked, complaint being made merely of "rheumatics." In all such cases it is advisable to make a physical examination of the nerves for the existence of tender points; for pain (the symptom) has, in the majority of instances, some objective basis. Examination will usually reveal the fact that the movements both of the arms and legs are limited owing to the pain which is thereby provoked. Gentle pressure will elicit tender points over the majority of the cutaneous branches of the nerves of the arms and legs, and usually also over the posterior spinal nerves. The patients, victims of this condition, are commonly women of middle age, and often at the climacteric period. It is also predisposed to by debilitating conditions, the chief of which is influenza, while worry and overwork are not without their causal influence.

Trigeminal Neuralgia (*syn., Tic douloureux; Prosopalgia*).—The pain in this condition may be limited to one or other of the anatomical divisions of the nerve, and is probably the most acute form of pain with which the physician has to deal. It is usually paroxysmal, coming on suddenly in exacerbations and lasting for a few moments, so that the severest form of this disease has received the name of epileptiform neuralgia. Most cases are marked by a distinct periodicity, but this is not now regarded as indicating a malarial causation. Various stimuli may excite this condition and induce a paroxysm. Thus the effect of cold is most marked, as also the movements of the jaw necessary in mastication. For this reason the facial expression of a victim of trigeminal neuralgia is characteristic; from the fear of starting an attack of pain, a stolid, emotionless appearance is maintained. The tongue is coated, sordes cover the teeth, and there is a fœtid odour of the breath, from the fact that the movements of the jaw and tongue necessary for the proper cleansing of the mouth invariably start a painful paroxysm.

Tender points may be obtained at all the foramina through which branches of the nerve issue upon the face, forehead, or temple. The eyeball also is an occasional seat of pain. A tender point may likewise be obtained in the ear; while a common seat of pain is to be found in the centre of the hard palate.

Various vasomotor effects are associated with this form of neuralgia; œdema over the side of the face subject to pain, localised sweating and flushing of the skin, and dilatation of the cutaneous vessels. Sometimes, also, convulsive twitching of the facial muscles on the side of the pain is observed (tic convulsif).

In the majority of the cases of trigeminal neuralgia no anatomical change is detected in the nerve or its sheath. Because a lesion is absent in many, it is not to be assumed that all such cases are of this nature. Pressure upon the branches of the fifth nerve by tumours—osseous or malignant—should not be overlooked. In one case, severe paroxysms of pain in the middle division arose from irritation of the nerve in the middle cranial fossa, as a result of a fall on the side of the head. In another, characteristic tic of the superior maxillary division was associated with disease in the antrum of Highmore; while in a third, unilateral supra-orbital pain of a neuralgic type was due to suppuration in the frontal sinus on the same side. There is no necessary relation between tic and disease of the teeth. Notwithstanding the extraction of the teeth in most cases of this nature, the painful paroxysms continue, often in an augmented form.

The theory has been advanced that true tic doulourеux is produced by a chronic degenerative process of the cells in the Gasserian ganglion. On this point, however, there is yet needed the corroboration of histological observation. The disease is essentially one of the decline of life.

Occipital neuralgia.—A condition characterised by pain at the back of the head and extending upwards towards the vertex, less intense and less paroxysmal than that of trigeminal neuralgia. The pain is not necessarily limited to the distribution of the great occipital nerve, but may be found also over the posterior branches of the cervical plexus. Hence tender points are obtained over the occipitalis major, where it penetrates the cervical fascia; on the scalp in the region of the parietal eminence; and over the trapezius muscle.

In this connection reference may be made to neuralgia of the scalp, a very painful and troublesome condition. Here there is general pain over the head, or a description is given as if the head were held in a vice. There is tenderness over all parts, but more marked where the nerve branches, be they of cervical or trigeminal origin, penetrate the fascia. The scalp is so tender that the mere act of brushing the hair is painful. It is usually associated with pain and tender points elsewhere, such as over the spine and in the arms; while the memory is often markedly and characteristically impaired. This condition is not limited to adult or late adult life, being commonly seen in adolescents of a neurotic disposition.

Brachialgia.—This is a troublesome and painful condition, interfering materially with the occupation of the sufferer. Complaint is made of intense pain in the arm, chiefly about the shoulder, and aggravated by movement. The pain is more or less constant with paroxysmal exacerbations, while it is especially severe and troublesome at night. A physical examination shows that passive movement of the arm is productive of as severe pain as is voluntary movement. Pain is elicited by pressure over the trunks of the nerves, as well as over the following points—(*a*) the branches of the circumflex nerve over the deltoid muscle. If the pain, as is not uncommonly the case, be limited to this position, a variety of brachialgia—deltoid neuralgia—is obtained; (*b*) the external cutaneous branch of the musculo-spiral nerve in the middle of the upper arm posteriorly; (*c*) the external cutaneous branch of the musculo-cutaneous nerve; (*d*) the palmar cutaneous branch of the median; and (*e*) the dorsal cutaneous branch of the ulnar nerve.

The causes of this affection are to be found in traumatism, rheumatism, and gout: while a painful condition of arthritic origin is seen in some cases of old standing hemiplegia, where the slightest attempt at movement gives rise to severe pain in the arm and shoulder. The majority of cases of so-called brachialgia are due to a perineuritis of one or more of the nerves of the brachial plexus. For details of the surgical procedures, nerve-stretching, neurectomy, neurotomy, and excision of the Gasserian ganglion, the reader is referred to works upon operative surgery. As regards such treatment, the general statement may be made, that whatever operation is adopted, relapses are prone to occur, except probably in the last, which has proved of more enduring value.

Intercostal and abdominal neuralgia.—A not uncommon seat of pain is along the course of the intercostal nerves, with tender points where the branches penetrate the fascia, namely, on either side of the vertebral column, in the mid-axillary line, and in front near the costo-sternal articulations. Respiratory and other movements of the chest usually increase the pain.

A common form of intercostal pain is that associated with herpes zoster; when this occurs in old people, it is often of great severity, very protracted, and difficult to treat. Corresponding to the distribution of these nerves are those of the abdominal wall, which may also, although less commonly, be the seat of neuralgia.

Sciatica.—Although probably in every case due to an inflammation of the sheath of the great sciatic nerve, this condition may be referred to under neuralgia. It is characterised by pain, of a constant, gnawing character, in the position of the trunk and branches of the sciatic nerve. If the nerve is affected throughout its course, the position of the pain is clearly indicated, and tender points are discerned—(*a*) over the situation of the great sciatic notch; (*b*) behind the great trochanter; (*c*) in the middle of the thigh posteriorly, and also in the popliteal space; (*d*) at a spot immediately behind the head of the fibula; (*e*) at the outer side of the ankle joint. In addition to these tender areas, pain may usually be elicited by pressure over the trunk of the nerve, at the back of the thigh, and over its external popliteal branch in the leg. Sciatica is almost invariably associated with sacrodynia; the pain is increased on movement, and is usually worse at night. In old standing cases associated with considerable inflammatory affection of the nerve sheath and subjacent nerve fibres, some degree of muscular wasting is observed, and impairment of the cutaneous sensation; but the knee-jerks are normal, and the electrical excitability of the muscles is not impaired.

A very troublesome form of neuralgia of the leg, is pain in the distribution of the anterior crural nerve. The pain is felt in the groin and down the anterior aspect of the thigh. It is usually associated with sciatica, but may be met with as an isolated condition. Tender points are to be detected in the middle of the groin and in the middle of the thigh anteriorly.

Rarer forms.—Pain of neuralgic character may also be met with in other parts of the body; such are scrotal and labial neuralgia; spinal neuralgia, affecting part or all the spine, from the cervical to the sacro-coccygeal region, and neuralgia of the penis.

Treatment.—For the alleviation of this painful and often protracted malady, the therapeutic resources of the physician are taxed to the utmost. For, in addition to the temporary relief of pain demanded by the patient, an attempt should be made to reach and cure the causal condition. Tic douloureux may be considered, first, as a type of the other varieties. The methods of treatment divide themselves into—

Remedies given with a view to cure; remedies administered locally and internally to relieve pain; the local application of electricity; surgical procedures—neurotomy, neurectomy, nerve stretching, and removal of the Gasserian ganglion.

(1) The etiology of each case should be separately considered; and for this purpose an examination of all the organs receiving nerve filaments from the fifth cranial pair should be made—the eyes, more especially with a view to the detection of errors of refraction; the nose, attention being directed to the condition of the turbinated bones and the antrum of Highmore; the teeth, for carious conditions, more especially those implicating the deeper lays of the dentine; the ears and the throat. Should any abnormal condition exist in these structures, their correction, surgically or otherwise, is the first consideration. A large number of cases exist in which no diseased state can be detected in these organs.

In many cases of trigeminal neuralgia of a non-malarial nature, quinine in doses of from 5 to 10 grs., given periodically, may produce a curative result. The administration of iodide of potassium in 10- to 15-gr. doses every four or six hours is often of marked service, in cases which have no syphilitic history, but in which the pain is probably due to thickening of the perineurium, where the nerve issues from a bony canal or fibrous ring. Should a distinctly rheumatic history be obtained, the combination of iodide of potassium with salicylate of soda (10 to 15 gr.) is often effectual. Some cases, but more especially the malarial and anæmic, may be improved, and eventually cured, by the administration of arsenic, either as Fowler's solution, or as the double iodide of mercury and arsenic, as in Donovan's solution.

(2) Of internal remedies for the relief of pain, mention will first be made of the so-called neuralgia specifics. Of these the chief are the following:—Probably the most serviceable is tincture of aconite given in doses of 1 minim hourly, and the effect watched. Tincture of gelsemium sempervirens, which may be given in doses of 10 minims every hour up to four hours. Butyl-chloral hydrate, 5 gr. every hour until 30 gr. have been taken. Actæa racemosa, in $\frac{1}{2}$- to 2-drm. doses of the fluid extract, or 30-minim doses of the tincture. Though not a "specific," as the previous remedies, it has been found equally useful. Bromidia in drachm doses is highly recommended by some, while the chloride and bromide of ammonium may also be of value. Cannabis indica, given in doses of $\frac{1}{8}$ to $\frac{1}{4}$ gr. of the extract, has been found useful by some. A host of synthetic remedies are now in the market for the relief of pain, many of which are valuable, some useless, and a few dangerous. Of those which may be safely recommended are:—Phenazone, in doses of 5 gr., every hour for three or four hours. Phenacetin, which may be administered in the same way as antipyrine. Exalgin, and less valuable antifebrin, in 2- to 3- gr. doses. As these remedies are, in addition to being analgesics, temperature depressors and blood destroyers, their continued administration should be watched with care.

A form of local medication, which has fallen lately into disuse, is often of marked service in relieving acute pain of a neuralgic nature. This is the hypodermic injection over the seat of pain, of substances having a local anæsthetic action. Such, for instance, is the hypodermic injection of osmic acid, in doses of one or two drops of the 1 per cent. solution; also of carbolic acid as a 2 per cent. solution; five to fifteen drops of chloroform; and of caffeine, in injections of approximately 1 gr. The local application of ointments may also be efficacious. Such are the ointments of aconite, veratrum, and menthol, or the liniments of aconite, belladonna, and chloroform.

But before resort is had to these applications, simple measures, such as hot or cold fomentations, mustard leaves (often most efficacious), or cantharides blisters should be tried.

One of the most distressing features of a severe case of trigeminal neuralgia lies in the difficulty of cleansing the teeth and mouth cavity, as well as the inability to masticate. These are due to the fact that movement of the jaw usually starts a severe paroxysm of pain. A certain amount of relief may be obtained by painting the inside of the mouth and gums with a 5 per cent. solution of cocaine, which causes a sufficient degree of anæsthesia to permit of the mouth being washed and food to be taken. There are many cases, however, which will not be materially affected by any of these remedies. In these the pain is usually severe and always paroxysmal—the so-called epileptiform neuralgia of Trousseau. In such cases the only remedy is to be found in the hypodermic injection of morphine. There seems to be no doubt that the prolonged administration of opium will bring about a cure in some cases; but the surgical procedures, to which resort is now more commonly had, should be recommended in preference.

In all cases, whatever their nature, too great attention cannot be paid to the general health. Iron, cod-liver oil, and alcohol, judiciously administered, are often productive of marked improvement.

(3) A useful, and in many cases a satisfactory method for the relief of trigeminal neuralgia is to be found in the local application of electricity. Of the two forms of electrical current, galvanism is that more commonly and more effectively used. Its method of application is simple. The negative electrode is placed over an indifferent part, while the positive is carried along the course of the pain, or put over the tender points of the nerve. Not too strong a current should be used; one sufficient to produce a feeling of tingling being all that is required. It will be found preferable to recommend this method for application by the patient himself. The application of the galvanism by a skilled person three or four times a day over a definite period is of little value. Much more satisfactory is the application of the positive pole to the area of pain as soon as a paroxysm commences. The sitting may be prolonged for four or five minutes, and often repeated.

In the treatment of brachialgia it is necessary to enjoin absolute and complete rest for the affected arm, which should, with this object in view, be placed in a sling, or bandaged to the side of the chest. So difficult is it at first to get a patient to appreciate the value of rest, that the cure is often indefinitely delayed. The second factor in the treatment is counter irritation. This may be employed either in its mild or severe forms, beginning with the application of mustard leaves over the tender points, up to the use of the actual cautery. Of internal remedies, the most satisfactory have been found to be a combination of the iodides and bromides with salicylate of soda. In the later stages, after the acute pain has passed away, tonic treatment by arsenic, iron, and quinine is recommended.

The treatment of neuralgia of the lower limbs should be conducted on the same principles as for the upper extremity. The first essential is rest in bed, and the second counter-irritation, either by blistering or dry-cupping. Should there exist an obvious diathetic tendency to rheumatism or gout, this should be counteracted by suitable remedies; but even in cases which show no such condition, the administration of the iodides and bromides, with or without the addition of such remedies as phenaectin and antipyrine, is valuable. Other sedatives may be employed, but in severe cases the hypodermic use of morphine is the only useful remedy. Cocaine, in doses of $\frac{1}{12}$ to $\frac{1}{3}$ gr., hypodermically is of much use in many cases; while chloroform (10 minims) and caffeine (1 gr.) may be similarly employed as an injection over the seat of pain. The local application of galvanism, in this as in other painful conditions, is often of great value.

W. ALDREN TURNER.

VISCERAL PAIN.

By this term is understood pain at certain points on the surface of the body produced by disease of underlying or adjacent viscera, which pain is usually accompanied by more or less definite areas of cutaneous tenderness. It may be said as a general principle, that each organ of the body stands in relation with one or more areas of the skin surface. When an organ is affected by disease, the skin area becomes objectively tender, and pain is referred to it. Hence such common symptoms as headache, pain in the back, infra-mammary pain, epigastric and interscapular pain, are found to be usually associated with areas of cutaneous tenderness and to be symptomatic of disease of adjacent viscera. By examination of these areas, the body may be marked out into a series of zones, and this has been done and the areas corresponding to the viscera localised by Head, to whose work the reader is more especially referred.

Symptoms.—The characteristic features of referred pains, by aid of which they may be distinguished from neuralgic pain, are briefly as follows:—The pain is associated with a disordered condition, temporary or otherwise, of the organ to which it is related; the pain is referred to a certain part of the area of cutaneous tenderness, which has received the name of maximum point; every area of cutaneous tenderness has one or more of these maxima; the maxima are fixed points, but do not correspond to the passage of a nerve either through a bony canal or fibrous ring.

The following may be taken to be a brief summary of the tender areas associated with visceral disease, so far as at present known. This has been taken from Head's work upon the subject. In affections of the eye, more especially those involving the refractive media and the ciliary muscle, the pain is referred to the frontal, temporal, and vertical scalp areas. In dental caries, areas of cutaneous tenderness may be found over the greater part of the forehead, nose, and temple, over the jaws, ear, and submaxillary region, and upper part of the neck.

Disease of the middle ear causes referred pain chiefly over the hyoid region, but sometimes also over the vertical and parietal areas of the scalp. Pain in disease of the tongue and tonsil is referred chiefly to the auricular submaxillary and chin regions.

In heart disease, the pain is referred along the first, second, third, and fourth dorsal areas; but at times it is more general, extending from the fifth cervical to the ninth dorsal segment. In pulmonary affections, the areas involved extend from and include the first to the seventh dorsal segments. In stricture of the œsophagus, pain and tenderness correspond to the distribution of the fifth dorsal segment. In disturbance of the stomach, the affected areas correspond to the seventh, eighth, and ninth dorsal segments.

In certain intestinal conditions the pain and cutaneous tenderness involve the ninth to the twelfth dorsal areas; and in those of the liver and gall bladder the eighth and ninth dorsal areas.

The kidney is more particularly associated with the tenth dorsal segment, and the ureter with the eleventh and twelfth. The testicle and epididymis are also associated with the like segments; to which segments the ovary also corresponds. The bladder corresponds mainly to the third and fourth sacral areas, the pain being referred to the perineum; while the pain and tenderness of prostatic disease are referred to the distribution of the fifth lumbar, and first, second, or third sacral segments; the pain extending in these cases over the ischial tubers, the calves and the soles of the feet to the great toe. The os and cervix uteri are related to the eleventh and twelfth dorsal and the third and fourth sacral areas.

Affections of the thoracic and abdominal organs may also be associated with tender areas over certain parts of the scalp, which fact may explain the frequent and well-known headache met with in various diseases of the organs of the chest and belly. Associated with these areas of scalp tenderness and pain are those of the body more especially related to the affected viscera.

W. ALDREN TURNER.

CONVULSIVE DISORDERS.

Under the term Eclampsia, or convulsive disorders, are included many conditions, differing from each other in causation, in the age at which they occur, in prognosis and in treatment; but all are characterised by the presence of more or less generalised muscular contractions or spasm. It is almost impossible to adopt a classification which will comprehensively embrace the whole series of convulsive disorders. Eclampsia is a symptom of many and diverse pathological conditions. For example convulsion is a symptom of disorders differing so much from each other as tumour, hæmorrhage, and softening of the brain; toxic states of the blood, resulting from renal disease, syphilis, or the presence of certain drugs; such simple conditions as cutting a tooth, a rise of temperature, or the presence of intestinal worms in neurotic children; and idiopathic epilepsy.

The following classification has been adopted, as it appears to the writer to be that which includes the greatest number of conditions of which eclampsia is a symptom:—Convulsions symptomatic of primary affection of (*a*) the nervous system, including partial epilepsy and general convulsions of organic nature: (*b*) the urinary system, embracing uræmic and puerperal convulsions. Infantile convulsions. Idiopathic epilepsy.

From Primary Disease of the Nervous System.

These may be, on the one hand, of a partial or localised nature, affecting merely one limb, a portion of a limb, or one side of the body: under which circumstances they have received the name of partial or epileptiform seizures—focal or Jacksonian epilepsy. On the other hand, universal convulsion, in no way differing from idiopathic epilepsy, except perhaps in the constancy of its local commencement, may arise from organic lesion, such as thrombosis of a cerebral vessel, or tumour involving the cerebral cortex.

JACKSONIAN EPILEPSY.

This is indicative of the existence of a localised source of irritation of the cortex cerebri or immediately subjacent tissue; the resulting phenomena being motor or sensory, according to the situation of the lesion.

Symptoms.—When motor, the march of the spasm is constant. Beginning either in one limb, or in the extremity of a limb, such as the hand or wrist, it tends to spread to other portions of the body, or remains limited to the limb originally affected. Thus, if the spasm commences in the face, it may spread to the arm and then to the leg; if in the hand, it may pass up the arm, then involve the face, and subsequently the leg. If commencing in the leg, it spreads to the arm and then to the face, A convulsion beginning in the face does not attack the leg and leave the arm unaffected. The local convulsions may, or may not, be accompanied by loss of consciousness; they may, or may not, become general; but they are invariably followed by a temporary paralysis in the parts or limbs convulsed.

The place of commencement of the spasm should be carefully noted, as it points to the portion of the cerebral cortex primarily or chiefly implicated. If the spasm involves the face, the angle of the mouth is the part in which it commences; if the arm, then it is commonly the thumb or the index-finger; while a curious instance of spasm from cortical irritation, involving principally the platysma myoides, has been recorded by Bramwell. Certain general directions regarding the localisation of a cortical lesion may be given as a result of numerous experimental and clinico-pathological observations. Thus—

(*a*) Spasm commencing in the thumb, fingers, or forearm, indicates a lesion involving the middle third of the ascending frontal and parietal gyri; (*b*) convulsion starting in the lower limb points to lesion of the upper third of the Rolandic region, the cortical area for which extends from the base of the first frontal gyrus, nearly as far back as the external parieto-occipital fissure; (*c*) convulsions starting in the face denote a lesion of the lower end of the ascending frontal gyrus; (*d*) spasm commencing in the tongue places the seat of discharge also at the lower end of the ascending frontal convolution, but anterior to the face movements.

But irritation may not be limited to the portions of the cerebral cortex subserving motion. Stimulation of the special sense areas may cause characteristic phenomena. Thus, irritation of the occipital region by a new growth has been known to produce visual hallucinations; of the first temporal convolution, sensations of sound; while a few rare cases of tumour, involving the tip of the temporo-sphenoidal lobe, have caused subjective sensations of smell and taste. Just as a local muscular spasm is followed by temporary paralysis, so an over-discharge from a sensory area is followed by temporary abolition of the special sense affected.

Cortical irritation may be produced by various conditions. Such are cicatrices, depressed fractures, or spicula of bone pressing upon the cerebral cortex; traumatic affections of, and new growths involving, the cortex and its membranes, either of a simple, malignant, or syphilitic character. So commonly, indeed, is the existence of focal epilepsy associated with syphilitic disease of the cortex or its membranes, that the term Jacksonian epilepsy is sometimes used synonymously with syphilitic cerebral disease of a convulsive nature.

As syphilomata arise most commonly from the meninges, they are found to occupy a superficial position. If discovered deeply in the cerebral substance, such as the centrum ovale, or basal ganglia, they have arisen from an ingrowth of pia arachnoid, or from the adventitia of a blood vessel.

The views regarding the explanation of localised convulsions, being largely hypothetical, need not be considered here. Their existence indicates irritation of cortical grey matter, whether from the presence of an actively growing tumour, the contraction of a cicatrix, or a diffuse gummatous condition of the pia arachnoid membrane. It has also been stated that convulsions may arise from a process of auto-intoxication, induced by the breaking up and absorption of gummatous products under treatment. Further, their production, as a result of sudden shutting off of the blood supply to a limited area of the brain cortex, should not be overlooked.

Convulsions arising from cortical irritation of organic nature are commonly tonic and clonic in character, and of short duration; but rare cases have been observed, in which rhythmical tremors have existed for days or even weeks.

Removal of the source of irritation, or of the portion of cortex subject to irritation, arrests the spasm. But this has not been found to be the case in athetosis, and choreiform movements of a post hemiplegic nature. The inference, therefore, is drawn, that the cause of such movements is to be found, not in the cortex itself, but towards the base of the brain, and more especially in the optic thalamus and posterior limb of the internal capsule.

But all fits beginning locally are not indicative of organic brain disease. It has been stated, when describing idiopathic epilepsy, that a not uncommon form of *petit mal* is characterised by the local incidence of the spasm. In this form, however, there is always, at some period, obscuration of consciousness; while, in the Jacksonian attack, the patient may watch, for example, spasm of the arm, during the whole of the seizure.

Treatment.—The treatment of partial epilepsy is determined by the cause. As this condition is in so many cases synonymous with syphilitic disease, vigorous antisyphilitic remedies should be adopted. The treatment of the other conditions, which may give rise to epileptiform attacks, should be conducted upon general principles.

General Convulsions from Organic Disease.

As already incidentally observed, convulsions of a more general nature, resembling epilepsy in their features, may arise from organic disease of the brain. Thus a child, who is the victim of infantile spastic hemiplegia due either to meningeal hæmorrhagic effusion, or plugging of a cerebral blood vessel, invariably at some time in later life develops convulsive seizures in no way differing from idiopathic epilepsy, except that the paralysed is usually earlier and more extensively convulsed than the non-paralysed side. To the condition of the brain which follows occlusion of a cerebral artery in infancy, the term porencephaly has been applied. This condition is characterised pathologically by great shrinking of the whole or part of the affected hemisphere; sometimes by the substitution of nervous tissue by cystic formation; in other cases, merely by diminution in size and sclerosis of some of the convolutions.

Hemiplegia following a vascular occlusion such as thrombosis of the middle cerebral artery in the adult, may be accompanied by periodic convulsive attacks affecting only, or chiefly, the paralysed side; and the sudden shutting off of the blood supply by vascular occlusion (embolism) may induce a convulsive seizure, which may or may not be repeated when hemiplegia is established.

Convulsive seizures, known by the term congestive attack, are common phenomena in many cases of general paralysis of the insane. In this condition they begin unilaterally, but may become general, are often of long duration, and are followed by deep coma, in which the patient may die. They are commonly due to an effusion of blood in the subdural space, a condition which pathologically has been termed — pachymeningitis hæmorrhagica.

To this list may also be added the convulsive seizures met with in tuberculous meningitis, hydrocephalus, and suppurative conditions of the cerebral meninges.

FROM DISEASE OF THE URINARY SYSTEM.

These constitute one of the manifestations of uræmia. The convulsive seizures in this condition resemble those described under idiopathic epilepsy. The onset may be sudden, or may be preceded for some hours, or a few days, by headache, giddiness, and drowsiness. There may be one or more convulsions of the epileptic type, with complete unconsciousness. The tongue is bitten, and the urine and fæces are passed in the bed. The convulsion usually commences with tonic, followed by clonic, spasm. In many cases the patient passes rapidly from one seizure into another, giving rise to the condition of *status epilepticus*, in which he dies. In this condition the temperature is raised, sometimes to hyperpyrexia.

The circumstances under which these convulsions occur are—acute and subacute inflammatory conditions of the kidney, and more rarely granular atrophy of chronic Bright's disease. During parturition, in cases with much albuminuria, the convulsions receive the name of puerperal eclampsia.

It would be a point of some interest to know how far the subjects of puerperal eclampsia have inherited a tendency to convulsions. It has been stated that some have a history of convulsions when teething, others at the onset of the pyrexia of acute infectious disease; and the statement has been made that eclampsia of this origin has been replaced by ordinary epileptic seizures (Féré). Hence it is probable that eclampsias of this nature occur in those with a neuropathic predisposition, and may form the starting-point of ordinary epilepsy.

W. ALDREN TURNER.

CONVULSIONS OF INFANCY AND CHILDHOOD.

The period from birth to puberty is characterised by a tendency to instability of the nervous system. Hence convulsions arise from causes which are insufficient or unlikely to produce eclampsia in adults. This instability, which is more or less natural to all children, is greater in those who inherit a disposition to the neuroses, and is fostered by the incidence of rickets.

The onset of acute infectious disease, associated with elevation of temperature, is a common cause of infantile convulsions, which may also be largely excited by peripheral irritation, such as teething, overloading of the stomach, diarrhœa, constipation, or the presence of intestinal worms, and indigestible food.

Infantile convulsions are manifested in various ways, sometimes as laryngismus stridulus, or as carpopedal contractions, while in a large proportion of cases the convulsions are of a mildly epileptic nature. Such symptoms are sufficiently grave, so long as they last, but they do not recur after the subsidence of the temperature, or the removal of the source of peripheral irritation. In other cases, however, the continuance of the convulsions is prolonged, to days, weeks, or months. Even when the duration is thus lengthened, some cases recover, but others develop into epilepsy. Hence in many cases of epilepsy the fits are stated to have commenced during infancy, while a history of teething fits is not at all uncommon in those who, towards the end of puberty, become epileptics.

But infantile convulsive attacks may, on the other hand, indicate the onset of serious organic disease of the nervous system; for instance, meningeal hæmorrhage, thrombosis in the substance or upon the surface of the brain, and acute anterior polio-myelitis. After the convulsions which occur at the onset of such an attack have subsided, loss of power in a limb or limbs, or on one side of the body, is observed; and the conditions known as infantile spinal paralysis and infantile hemiplegia are found to have been established.

The future of a child, the victim of infantile hemiplegia, is serious. It is invariably associated with defective development of the paralysed side; often with a state of dementia, amounting in some cases to idiocy; and in the majority of cases, about two-thirds of the total number, with epilepsy.

In early childhood, as in infancy, some of the same causes are at work in the production of convulsions. Peripheral irritation, however, becomes a less potent factor, while there is introduced the effect of shock, fright, or sudden mental emotion. In many cases of epilepsy no other excitant is obtained than the influence of fright in childhood. It is, however, unlikely that this cause alone is sufficient to produce epilepsy, apart from an inherited instability of the nervous system, or a tendency to the neuroses.

W. ALDREN TURNER.

EPILEPSY.

Epilepsy is a paroxysmal convulsive disorder of greater or less intensity, the characteristic feature of which is a sudden loss or impairment of consciousness, which may or may not be preceded by a warning, and is not necessarily due to any obvious organic disease of the brain or its coverings.

The feature whose presence is essential to establish the diagnosis of epilepsy may consist of any degree of unconsciousness, from a momentary blunting up to complete abolition of conscious sensation. The convulsive element is not necessary for the establishment of epilepsy: it may be entirely absent, or it may exist to any extent, from a slight involuntary turning of the eyes up to a universal spasm.

But while the term epilepsy is given to such a condition, arising from no known gross lesion, there are several forms of organic brain disease which occasion symptoms of a convulsive nature, differing little if at all from those of idiopathic epilepsy. Of these may be mentioned chronic irritative conditions arising from thrombosis of the cerebral blood vessels, with secondary softening, chronic meningo-encephalitis, old-standing foci of hæmorrhage, and depressed spicula of bone, and intracranial new growths.

Etiology.—**Prevalency.**—Concerning the general prevalency of epilepsy, in its relation to the total population, there is difficulty, from many causes, in arriving at satisfactory statistical results. First, no census for this purpose has been taken in the United Kingdom, while those made abroad have been only partial; and, secondly, epilepsy is so closely related in the early years of life to idiocy and imbecility, and in the later years to insanity, that it is almost impossible to satisfactorily separate them for statistical purposes. But a few facts may be given upon the estimated number of adult epileptics. In Germany the estimate is given as 1 per 1000, in New York State as 2 per 1000, and in the Canton Aargau in Switzerland, as 2·5 per 1000 of total population. In Italy, of 10,000 young men who presented themselves for military duties, 11·5 were rejected owing to epilepsy, giving a proportion of just over 1 per 1000. It would not, therefore, be placing the number too high if it is stated that the probable proportion of adult epileptics to the total population averages from 1 to 1·5 per 1000 in the European countries. From a census upon this subject, taken in the Australian colonies, it was found that the proportion was lower than that estimated for Europe and America. In the colony of Victoria, for example, it is stated that there is one epileptic in every 2715 of population, or, roughly, an average of ·25 per mille. These estimates have been taken from all cases of epilepsy, whether met with inside or outside of asylums. It would not, therefore, be putting the proportion too high when it is stated that in Great Britain there is one epileptic in every thousand of the population outside the asylums.

Heredity.—The influence of heredity in the production of idiopathic epilepsy is a subject of great importance. It is not essential that epilepsy should have existed in the parents or progenitors of an epileptic person, but merely a tendency to one of the neuroses, such as insanity, alcoholism, or hysteria; while stress has been laid by some authors upon the influence of chorea and somnambulism in the ancestry. The statistical results of several authorities, who have studied the question of similar heredity, approximate with a sufficient amount of uniformity to be of value, although this influence has been minimised by some. Reynolds found a hereditary predisposition in 31 per cent. of his cases, Gowers in 35 per cent., Berger in 32 per cent., and Echeverria in 27 per cent. Gowers and other writers have stated that, when an inherited tendency exists, the female members of the family suffer more frequently than the males. A fact of some statistical interest has been mentioned by Bouchet, to show the relation of parental epilepsy upon the incidence of convulsions in the offspring; of fifty-eight children, born of fourteen epileptic mothers, thirty-seven died young from convulsions, and of the remaining twenty-one, seven became epileptic. The relation between phthisis and epilepsy, though common, seems to be rather of the nature of a coincidence than to point to any direct predisposing effect of the former in the causation of the latter.

Age.—Of the etiological factors, the age of the individual in relation to the incidence of epilepsy is that most clearly determined by statistics. The collections of all authors, who have dealt with large numbers of epileptics, show that the commonest period for the commencement of epileptic fits is between the ages of 10 and 20 years. Thus, of 1450 cases collected by Gowers, 39 per cent. commenced during this period; of 995 cases collected by Hasse, 36 per cent.; and of 980 cases by M'Lane Hamilton, 27 per cent. In Gowers' cases this contrasted with 28 per cent. in the first decade, 14 per cent. in the third, and 5 per cent. in the fourth decades. During the second decade, the years most fruitful for the incidence of epilepsy are from 14 to 17 inclusive. Reynolds pointed out that when there was a marked hereditary influence, the disease began somewhat earlier than in those in which no such history was obtained.

Exciting causes.—As the existence of a similar heredity towards epilepsy is found in only one-third of the total number of cases, so likewise it is with the presence of exciting causes. In from two-thirds to one-half of the total number of epileptics, no satisfactory excitant is obtained, although many conjectural circumstances are narrated by the patients or their friends. Hence it is necessary to exercise discretion in their selection. Of the causes usually assigned, the commonest appear to be mental emotion, such as worry, fright, and prolonged anxiety.

The influence of traumatism, such as blows, falls, or kicks upon the head, is probably not great in the production of true epilepsy. Much more commonly does injury lead to convulsions of the Jacksonian type, from local injury to a part or parts of the brain. But whether the cerebral injury is due directly to traumatic causes, or arises from intracranial disturbance, such as is productive of infantile hemiplegia, convulsions may eventually ensue, indistinguishable from those of true epilepsy except in their local commencement, and the tendency to affect one side of the body more than the other.

The influence of the exanthemata (scarlet fever and measles) and of typhoid fever should be borne in mind in the causation of epilepsy. Of those, scarlatina is the commonest cause, and this apart from nephritis or cerebral thrombosis arising from middle ear disease.

The influence of syphilis as a factor in the production of true epilepsy requires further investigation. The acquired form of the disease occasions convulsions of a typically Jacksonian type, from organic lesions of a characteristic kind; and the injury to the brain, thus produced, may in some cases give rise to fits indistinguishable from true epilepsy. The evidence as to whether epilepsy may be due to the blood state in the so-called secondary stage of syphilis, as stated by Fournier, is extremely meagre. That epilepsy arises in the subjects of inherited syphilis is well known; but it is more reasonable to believe that the fits are evidence of a lowered nervous vitality, rather than a direct effect of the syphilitic condition.

It is improbable that masturbation has any direct influence in the causation of idiopathic epilepsy; it is more likely that the habit of self-abuse when carried to excess, and the convulsive seizures, are due to the same inherited neurosis.

The influence of alcohol in the causation of epilepsy requires mention in this place. It is probably one of the commonest factors in the development of epilepsy late in life. It is difficult, however, to obtain reliable statistics on this point. One observer (Fürstner) found that about one-third (31 per cent.) of his cases of delirium tremens were epileptic; while another (Moeli) found a larger percentage, namely, 36·4 per cent.; though in the alcoholic insane only 10 per cent. were epileptic. It is not, therefore, clear whether, in some cases, the alcoholic tendency and epilepsy may not be the results of the same inherited neurosis, rather than stand in the relation of cause and effect. Alcohol taken freely is undoubtedly detrimental to epileptics.

Pathology.—The naked-eye examination of the brains of epileptics is, as a rule, negative. No constant pathological lesion has been detected, although various conditions of no intrinsic value, such as thickening of the bones of the skull or of the membranes, and sclerosis of the cornu ammonis, have from time to time been described. If death has taken place during a fit, or in the status epilepticus, extensive engorgement of the cerebral sinuses is detected, and minutie hæmorrhagic ecchymoses may be discovered.

Notwithstanding the advances made within recent years into the minute histology of the nerve cell, the morbid change, if any, underlying the epileptic condition is still undescribed. Various changes, met with in epileptic brains, have been detected; the most prominent being that described by Bevan Lewis in cases of epileptic insanity, namely, fatty alteration in the nuclei of the cells of the second cortical layer, with vacuolation and disintegration of the cell protoplasm.

The facts of experimental physiology clearly point to the cortex of the cerebrum as that region from which the discharge takes place; but what is the cause of a periodical hyperexcitability of this region, or whether any change other than a hypothetical molecular one occurs in the nerve cells, is at present unknown. The locality from which the discharge originates is indicated by the symptoms, or phenomena, which usher in an attack. From this spot the discharge spreads, so that, in a severe epileptic seizure, the whole muscular system of the body is thrown into a state of spasm.

In cases characterised only by loss, or impairment, of consciousness without convulsion, or in those in which sudden loss of consciousness is the first symptom, and immediately precedes the convulsion, it is not unnatural to suppose that the part of the cerebral cortex especially related to the mental processes is that only, or primarily, involved. In like manner, in those cases beginning with a sensory warning, the argument has been advanced, that the discharge originates in a special sense cortical area; and that this is probably so, is gathered from the few cases submitted to pathological examination, in which a tumour implicating certain areas of special sensation gave rise to convulsions preceded by auræ of special sense. That those fits beginning with a definite movement of a limb originate in the cortical motor zone, in which the extremity is represented, is now one of the best established facts of cerebral pathology.

Symptoms.—For purposes of description it has been found advantageous to divide the several forms in which epilepsy manifests itself, into three groups. *Le petit mal*, characterised by momentary loss, or blurring, of consciousness, without evident muscular spasm. In this group epileptic vertigo finds a place. *Le petit mal with slight convulsions*—the epilepsia mitior with evident spasm of Reynolds. *Le grand mal*, characterised by complete loss of consciousness and universal convulsion.

Le petit mal.—A momentary loss of consciousness without evident muscular spasm. Individuals suffering from this condition often complain of "fainting attacks," or attacks in which they feel "dazed," or of "sensations," or of temporary loss of memory; or the friends may give an account of attacks in which the patient "looks queer." These seizures may last from one to three or five seconds. If the person is in conversation he suddenly stops speaking, his face becomes pale, and this may be followed by a slight flush; the pupils often dilate, and the expression assumes a "blank" appearance; these rapidly pass away, and conversation is resumed as if nothing had happened. If there is absolute, though momentary, abolition of consciousness, the patient may be noticed to stagger slightly, or lean to one side; but he rarely falls, or lets an object drop from his hand, and if walking, may go on doing so without faltering.

Usually such attacks give no warning of their approach, and they may pass off without any dulling of the mind. On the other hand, the loss or obscuration of consciousness may be preceded by momentary giddiness, and may be followed by temporary blunting of intelligence, or by acts of an involuntary automatic nature. Hence it is in association more especially with attacks of *le petit mal*, that the extraordinary actions known as epileptic automatism are met. For instance, one patient sometimes turned out the contents of his pockets; while on other occasions he has been observed to pile the crockery on the dinner table. Another invariably took out his watch without knowing what he did; while a very common automatic act, both after slight as well as severe fits, is the habit of undressing.

A form of *petit mal* is manifested in the so-called epileptic vertigo, a symptom which in itself indicates an interference with, although not necessarily a loss of, consciousness. It is paroxysmal in its onset, and is usually accompanied by nausea. There may or may not be slight convulsion associated with it. Vertigo or giddiness is, on the other hand, a common warning of severe epileptic convulsions.

The importance of the proper diagnosis of such slight attacks as those just described, cannot be over-rated. A by no means uncommon history is that of a person who has been suddenly seized with a severe epileptic fit while at work; inquiry into the previous history brings out the fact that for some time, it may be many years, he has been subject to temporary obscurations of consciousness. Or again, a patient, who is the victim of occasional attacks of *le grand mal*, may, during the intervals of these seizures, have numerous and frequent attacks of *le petit mal.*

Le petit mal with slight convulsions, the epilepsia mitior with evident spasm of Reynolds, is not at all an uncommon form of epilepsy; and it is one in which, owing in many cases to the local nature of the spasm, the diagnosis may at the outset present some difficulty. The convulsion, however, in whatever locality, or to whatever degree, is always associated with loss, or obscuration, of consciousness of a temporary nature. There may be merely a momentary conjugate deviation of the eyes, or of the head and eyes to one side; or a slight drawing over of the angle of the mouth; or an arm may be thrown into slight tonic convulsions of flexion, pronation, or supination; or the convulsion may take the form of a jerking of the arm and leg upon one side, clonic convulsion. Hence in this variety the convulsion may be either tonic or clonic. In the severer forms the clonic succeeds the tonic spasm. In some cases a distinct sensation, as if the limb were being moved, may precede or take the place of the actual convulsion. The patient is often aware of the commencement of the attack, though having no power to arrest it. Such attacks are only of momentary duration, several may occur in close sequence, and the total number recorded in one day may be very large.

It would appear as if this form of epilepsy was commoner in children and young people. When it occurs during sleep it is a frequent cause of "wetting the bed." Owing to the temporary duration and slight nature of the convulsion, the tongue is rarely bitten.

Le grand mal.—This common form of epilepsy is characterised by sudden and complete loss of consciousness, accompanied by violent muscular convulsions.

In considering the symptoms which precede an epileptic seizure, it is necessary to distinguish those which occur some days or hours before the attack from those which immediately usher it in. The former may be regarded as prodromal or precursory symptoms, while the latter receive the designation of warnings or auræ.

The premonitory symptoms vary largely; in some cases they are entirely absent; in others, they assume the form of changes in the mental condition. Thus, those who live with epileptics often tell precisely when the individual is likely to have a fit or batch of fits. On the one hand, some epileptics show abnormal moroseness, and a morbid irritability leading to frequent quarrels with their fellows, with whom as a rule they live on terms of amity; or a tendency to wander about in a depressed or melancholic fashion. Others, on the contrary, show a liveliness and sense of wellbeing, out of all proportion to the circumstances; while in others, again, distinct delusions may give indication of the approach of the fit. More purely subjective symptoms may take the place of the mental phenomena just described, namely, headache, giddiness, and loss of appetite.

The warnings or auræ of epileptics are legion; and it would serve no useful purpose to enumerate them. The clinical value of the aura, on the other hand, is great, and more especially if the warning is a sensation, or a movement of local nature, for an indication is thereby given of the particular region of the brain at which the epileptic discharge commences. Hence the value of investigating the aura of all cases beginning locally. Such local commencement, more especially if it be in the arm, leg, or face, is commoner in organic brain disease, but is by no means unknown in idiopathic epilepsy, and more especially in the form described as *le petit mal* with evident spasm. Auræ involving the special senses are likewise of importance, indicating the commencement of the discharge in a special sense cortical area. There are a few rare cases on record in which a tumour of the brain, involving the cortical areas for taste and smell, occasioned a special sense aura associated with a dreamy state. Various auræ of a more general nature are usually described by epileptics. A very frequent one is an epigastric sensation; while cephalic sensations and more especially vertigo are common.

In rare cases the fit is ushered in by a co-ordinated movement, and most writers on epilepsy record curious instances of such conditions; for example, one patient ran rapidly round the table and suddenly fell down unconscious; another would stoop down and peep under the sofa (Reynolds); while Gowers mentions the case of a patient who turned round and retraced his steps before the attack. To this variety the name epilepsia cursiva has been given. In a large number of cases, one-half according to Gowers, there is no aura, the loss of consciousness occurring so suddenly that the patient is unaware of the onset of the fit.

Three stages, fusing imperceptibly with each other, may be described as characteristic of *le grand mal.*

The stage of tonic convulsion, in which there is absolute loss of consciousness; tonic contraction of all the voluntary muscles, but usually more marked on one side than the other, so that the head and eyes are deviated, with torsion of the body and perhaps actual rotation; rigidity of the limbs; fixation of the chest by spasm of the intercostal muscles and diaphragm, which may be accompanied by a groaning sound of characteristic nature, forming the chief variety of the so-called epileptic cry; pallor of the face, followed by a dull red, congested appearance; dilatation of the pupil, and probably diminution in the force of the pulse. After lasting for approximately half a minute, this stage gives place to that of clonic convulsion. In this the muscles are jerked violently; the tongue may be protruded between the teeth and bitten, the bladder and rectum may be evacuated, and the eyeballs rotated in all directions: respiration is impeded by the irregular action of the respiratory muscles; the patient foams at the mouth, and the face becomes livid; and the veins of the neck are engorged. Just when the fit appears to be at its worst, and death from asphyxia seems imminent, a slackening in severity occurs, and the third stage is entered. In this a return to consciousness is the first symptom. The involuntary convulsive movements give place to those of a more voluntary character. Thus the patient may attempt to alter his position by turning round, or by sitting up. On the other hand, movements of a distinctly hysterioid nature may ensue, such as laughing, crying, or singing; or, again, he may struggle and fight violently, requiring several attendants to hold him down. All these, however, pass off, and the patient falls into a deep sleep or even state of coma.

The duration of the epileptic seizure is a point of some importance. The first stage may be said to last from five or ten seconds to half a minute. The second stage from one to two minutes; while the third is of varying duration according to the nature of the movements, being sometimes prolonged to eight or ten minutes. The post-convulsive sleep may last for an hour or two, and sometimes the patient is sleepy and dazed for the remainder of the day. Some points noted during and after the seizure require more detailed consideration.

The pupil as a rule dilates with the onset of the convulsion, and remains so during the stage of asphyxia. A preliminary contraction has been observed in some cases, and Reynolds pointed out the alternate contraction and dilatation which is observed for a short time after the fit is over.

The pulse also shows certain modifications; as a rule there is no initial alteration, but during the second stage it becomes full and bounding, and increased in frequency.

An epileptic fit is usually followed by severe headache, which is noticed when the patient wakes up from the post-convulsive sleep. The condition of the reflexes is of some importance, as a means of distinguishing the post-epileptic coma from that due to other causes. Immediately after the fit, the reflexes, both plantar and knee-jerk, are abolished (Westphal): but this is followed by a stage of increase, in which the knee-jerks are exaggerated and an ankle-clonus may be obtained.

The temperature may be slightly raised — from 1° to 2° F. — by the epileptic convulsion. In the status epilepticus to be presently described, whether met with in idiopathic epilepsy, or as a congestive attack in general paralysis of the insane, the temperature may rise to hyperpyrexia —106° F., or over.

Vomiting may follow an epileptic fit; in some it is an invariable sequence. When it occurs it forms an element of danger, owing to the risk of choking from food entering the larynx. The secretion of urine is copious, but in other respects it does not differ from normal. An epileptic rarely dies as a direct result of a fit; death may occur accidentally, either from suffocation, falling into fire or water, or from injury to the head received while falling.

Onset and frequency of epileptic fits.—As already pointed out, idiopathic epilepsy commences most commonly during the second decade, and especially during puberty, from the ages of 14 to 17 inclusive. The mode of onset is variable. In some cases, attacks of *le petit mal* may precede for years the onset of a severe fit; in others, one severe fit may occur without repetition, for periods varying from a few months to as many years; or, again, the first seizure may be followed regularly at intervals of a few weeks or a month or two, by similar single attacks, or batches of fits; or, the onset may be in the form of a series of fits, which is followed by an interval. As already pointed out, intermittent "wetting of the bed" is highly suggestive of fits occurring during sleep.

The onset of epilepsy is often ascribed to convulsions during teething. Many epileptics have indeed suffered from these convulsions, but there is no necessary relation between them, for often an interval of many years elapses between the teething fits and the onset of true epilepsy. Both, rather, are due to an inherited neurosis, which finds expression in convulsions. Of epileptic fits beginning in infancy, on the other hand, many follow directly upon convulsions arising during dentition.

Fits vary enormously in frequency; from one seizure once or twice a year, or less, up to several score in one day. They occur both during waking and during sleep. A common time for their appearance is the period just before or just after rising in the morning. Some epileptics only have fits during sleep; and this both at night, and if they, perchance, fall asleep during the day. Epileptics having only nocturnal attacks have been known to remain ignorant of their disease for years.

There are certain times in the sexual history of women, when they are apparently prone to the incidence of epilepsy, *e.g.* fits seem to be commoner at, or just before, the menstrual periods, and it is a matter of common observation that the onset of epilepsy in girls is often associated with delay or difficulty in the catamenia. In some women predisposed to epilepsy, lactation may form an exciting cause for the development of fits. One patient only had attacks during the period she was suckling, and this occurred after the birth of three successive children. Weaning the child on each occasion at once arrested the fits. The relation of epilepsy and pregnancy requires further observation. In one case, the fits entirely ceased from the time of quickening to the birth of the child; in another, the frequency of the fits was increased during pregnancy; in a third, they were diminished in number. In a fourth case, the first fit came on at the time of quickening, a circumstance which again happened at quickening in a succeeding pregnancy.

Epileptic fits occur both singly and in batches. Usually, during the interval, the patient is free both from sensations and subjective symptoms, and in many instances is perfectly capable of carrying on his work or profession. At times, however, the fits follow each other with such rapidity that a condition of acute dementia may be temporarily induced. In others, again, the condition known as the status epilepticus occurs, in which, during the coma following a series of rapidly recurring fits, convulsions take place, so as to reduce the patient to a condition of great danger, and death not infrequently ensues.

There is little to be obtained of value from studying the periodicity of epileptic fits. At the Epileptic Colony it has been found that by far the commonest time for the onset of fits was in the first hour after getting up in the morning; fits were rare on going to bed, and that as a rule the patients were much better and brighter in the evening. There were considerably more fits during waking than during sleeping hours, and more also in the house than outside; while there was a general impression that more fits occurred in warm and close weather than in cold.

The mental condition in epilepsy.—The mental state of epileptic persons is an important feature of the disease. In this connection there has to be considered the effect produced upon the mind by the continued occurrence of epileptic seizures over a number of years, as well as the attacks of acute insanity which replace, or follow, one or more fits. The mental phenomena met with in epileptics may be considered under the following divisions:—

1. *The association of epilepsy with idiocy or imbecility*, these being, on the one hand, directly the result of epileptic fits arising during infancy; or, on the other hand, both are the result of an inherited neurotic condition. Cases of this nature are among the most difficult with which the physician has to deal. The patients are invariably dirty in habits, usually destructive, and always irritable. They can only be treated satisfactorily in the idiot asylums.

2. *The interparoxysmal mental symptoms.*—These phenomena chiefly call for description in this place. There seems to be no doubt that the constant recurrence of epileptic fits over many years leads, in the majority of cases, to mental impairment and deterioration of a characteristic nature. In some cases this is slight, consisting only of a blunting of the memory and a dulling of the finer feelings and emotions. In other cases, a more marked and deeper degree of dementia exists, characterised by marked loss of memory, a slowness of perception and of appreciation of ordinary circumstances; an inability to originate, or to effectually carry out work, unless under direction; in another set, hypochondriasis, merging at times into actual delusional insanity, not necessarily of a constant type; in others, irritability and impulsiveness to an abnormal extent; while nearly all epileptics show a curious and altogether morbid religious emotionalism.

In some cases, as already stated, the onset of epilepsy, or of individual fits or batches of fits, is observed to be preceded for some days, weeks, or even months, by mental dulling and stupidity; or by hypochondriacal feelings, or by an unusual sense of well-being, or by unwonted contrariness, or by loss of memory, and sometimes delusions, which disappear when the fits ensue, or the patient is brought under the influence of the bromides. Clouston refers to a few rare cases, in which a chronic form of insanity took the place of fits which stopped at the menopause.

3. We have already stated that an epileptic fit is not uncommonly followed by symptoms, sometimes of an hysterical, at others of an automatic nature. Hence it is not difficult to realise that, in some epileptics, acute mental symptoms may follow an epileptic seizure. Of this nature, the most difficult to deal with on account of the intensity of the excitement, is acute epileptic mania, a condition in which the person may become homi-

cidal or suicidal; or subject to paroxysms of extraordinary fury and violence, requiring restraint. It is not unlikely that such forms of mania, exemplifying various states of defective inhibition, may actually replace epileptic fits in some persons; when this occurs the condition has received the name of masked epilepsy.

At the opposite pole from this condition is the acute epileptie dementia, sometimes seen after a batch of severe fits. In this state the patient lies in a semiconscious state, requiring to be fed and nursed; after a few days he begins to show response to external conditions, and is often subject to delusions, but usually returns to the state of health present before the seizure.

The physical condition of epileptics requires brief consideration. And, first, the *facies epileptica* should be mentioned. It is difficult to describe, but easily recognised by those who are in constant association with epileptics. In many the circulation is slow, and the extremities blue and cold. Constipation is a marked feature in most cases, and the appetite is sometimes enormous. Headache and giddiness are symptoms of which the epileptic often complains. Tremor of the muscles, chiefly clonic spasm, is sometimes present. The knee-jerks are usually active.

Diagnosis.—The diagnosis of epilepsy is in many cases simple, in others it is a matter of considerable difficulty. An affirmative answer to one of the three following questions is usually sufficient to establish the diagnosis. During the attack—Did the patient fall down unconscious? Was the tongue bitten? Was urine passed involuntarily? But a negative reply to these questions does not put the possibility of epilepsy out of court. None of these features are found in the variety known as *le petit mal* without spasm, or in the so-called epileptic automatism. For the diagnosis of these conditions reliance should be placed chiefly upon a consideration of all the facts of the case. Automatism is diagnostic of the epileptic nature of an attack; but it is often difficult to differentiate between *le petit mal* and a "faint" of syncopal nature. The chief diagnostic point lies in the suddenness both of the onset of, and recovery from, unconsciousness, in "faints" of epileptic character.

Even bearing such points in mind, it is not easy, in many cases, more especially if a fit is being described rather than seen, to differentiate those which are epileptic from those of hysterical nature. The fact that many fits are epileptic at the commencement, and terminate in hysterical manifestations, tends to confuse the diagnosis, as the epileptic phenomena may be brief and largely obscured by the hysterical. Hence the value of noting carefully the existence of an aura and the exact mode of commencement of a convulsive seizure. Moreover, the incidence of hysterical symptoms, following upon an epileptic attack, is not confined to the female sex, many male epileptics showing marked post-epileptic hysteria. But the following points, positive and negative, may be stated as being characteristic of an hysterical attack, in contra-distinction to epilepsy:—

1. Tongue-biting never occurs, but other parts of the body may be bitten as well as onlookers; involuntary micturition is unknown.

2. General struggling, throwing about of the body, kicking, fighting, etc., instead of the regular tonic, followed by clonic, convulsion of the epileptic.

3. The duration of the hysterical seizure may extend from a few minutes to a much longer time; the epileptic convulsion never exceeds a few minutes.

4. The epileptic fit terminates in deep sleep, the hysterical seizure at most in a dazed condition.

In the detailed description of epilepsy already given, reference has been made to the difficulty of distinguishing one form of epileptic fit—*le petit mal* with slight convulsion—from attacks due to organic brain disease; in both kinds the attack has a local commencement. The existence of loss, or obscuration of consciousness, has been already mentioned as the characteristic feature of epilepsy; while its association with minor attacks, due to organic brain disease, is rare. Should unconsciousness, however, be present in this condition, other evidence of intracranial new growth will probably be found.

A form of epileptic seizure may also occur, from irritation caused by an old hemorrhagic focus, or softening arising from blocking of a cerebral blood vessel. These attacks have all the features common to idiopathic epilepsy; but they are readily distinguished from this condition by the co-existence of hemiplegia. In this condition the attacks usually have a local commencement, and the paralysed side is more convulsed than the non-paralysed. The commonest form of this is seen in the cases of so-called infantile spastic hemiplegia, which is usually associated with considerable blunting of the mental faculties; but it is also met with in old hemiplegics.

Prognosis.—A person rarely dies in an epileptic fit; death, however, may occur from accident or injuries received during the state of unconsciousness. Thus, an epileptic may be suffocated during a seizure, or may fall into water, or into the fire, or from a height. Whether epilepsy may be cured by prolonged administration of drugs is a point not easy to settle. In most cases the constant administration of the bromides has kept down the fits, but in many such instances arrest in the use of the drug occasions a recrudescence of the seizures.

The vast majority of epilepties tend in course of time to exhibit some mental impairment. On the other hand, there are cases of epilepsy which undergo cure, occurring in young people, without any marked heredity, but in which an exciting cause, such as scarlet fever, is given. Night fits seem to be less susceptible to treatment than those which occur in the day; while attacks of *petit mal* are less influenced by drugs than the severer convulsive variety.

Treatment.—**Education and general management.**—Apart from the actual necessity of lessening the severity or diminishing the frequency of epileptic fits, by the administration of suitable drugs, other important measures in the general treatment of epilepsy ought to receive attention, and more especially the care, education, and management of sane epileptics.

In the first place, the care and education of epileptic children will be considered. For this purpose the sane should be separated from the imbecile or idiotic. For the management of the latter, provision is made in the idiot asylums. But it is more particularly to the education of the former class to which attention is directed. In the majority of instances these persons are kept at home, and the reasons for this are many and obvious. Hence the education of the sane epileptic child is disregarded; as he grows older, owing to inability to obtain a livelihood, he becomes a burden to his relatives, added to which is the natural tendency towards blunting of the intellect which epilepsy produces, and he eventually drifts either into an asylum or the workhouse.

Most medical authorities are agreed that no harm results from mixing together at school the epileptic and non-epileptic. But the fact exists that the presence of an epileptic has a disturbing influence upon his schoolfellows, as well as from the fact that the epileptic child requires special supervision. Although many epileptic children are bright and ready to acquire knowledge, the minds of a large number have, even at an early age, suffered from the blunting influence of their disease. Under such circumstances, it is on the whole better that they should be educated apart from healthy children; and for this purpose the following suggestions may be made:—(*a*) That where possible, and when there is no blunting of the intellect, or when the fits only occur at night or at long intervals, epileptics may be educated along with healthy children; (*b*) that if there is no marked impairment of intellect, but the fits are frequent and severe, or special supervision is necessary, or for other like reasons, provision should be made for the education of such children separately from others; and (*c*) that if along with epilepsy there is feeble-mindedness, without actual idiocy or imbecility, provision for suitable education should be made, and preferably in colonies, to be presently mentioned.

As regards the general management of the adult epileptic, many are perfectly able to conduct business and to carry on work of an intellectual or mechanical nature with credit and without any harmful result. On the other hand, either from gradual impairment of the mental faculties, or from inability to obtain regular employment owing to their disease, many epileptics are unable to make a living. Hence, in all classes of society, the epileptic, whose fits debar him from useful occupation, becomes a burden to his relatives; while amongst the poorer classes he swells the ranks of the unemployed, and eventually ends in the workhouse. It is in order to mitigate this evil that *colonies* where epileptics may be put to work under care and supervision, and at the same time receive medical treatment, are being developed. The principles underlying such treatment are—removal of the epileptic from the towns to the country; regular employment of an outdoor nature, under the supervision of capable persons; the maintenance of a well-ordered and regular mode of life, with avoidance of excitement, and abstinence from alcoholic liquor; and abundance of good nourishment of a simple nature.

The effect of such treatment has been found to be highly beneficial. The fits are diminished in number, as well as in severity, in many cases; while the general physical condition shows marked improvement.[1]

Medicinal treatment.—Since the introduction of bromide of potassium by Locock in 1857, in the treatment of epilepsy, this remedy and its analogues—the combinations of bromine with the other alkaline earths—have been used, not only to diminish the frequency and severity of the attacks, but also to prevent their recurrence. Of the bromine salts, bromide of potassium is that most commonly used and of the greatest service; but a combination of the bromides of potassium, sodium, and ammonium is of value in many cases. The bromides may be regarded as specifics in the treatment of epilepsy. By far the largest number of cases do well under these salts; in some, the disease has been cured; in others, they have so far modified the frequency and severity of the fits as to permit of the patient doing work; while in quite a minority they have no obvious influence upon the disease. In the cases in which this drug is of value, any break in its administration, however temporary, usually leads to a recrudescence of the fits. In addition to the effect of bromide upon the fits, it is also of influence in clearing up the mental condition commonly associated therewith. There are diverse ways of administering bromides. Some physicians prefer to give frequently repeated small doses, from 10 to 15 or 20 gr. thrice daily, or night and morning. On the other hand, one large dose at bedtime of a half to 1 drm. is often of more value, more especially in the case of nocturnal and matutinal fits. Bromide is best administered about one hour after a meal. Children bear the drug well. The administration of large and oft-repeated doses of bromide is to be deprecated, as experience shows that many epilepties may be rendered mentally stupid by this remedy.

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[1] In England the colonies for epileptics are:—(1) The colony at Chalfont St. Peter, Bucks, the property of the National Society for the Employment of Epileptics; (2) Maghull Epileptic Colony, near Liverpool; (3) The Meath Home, Godalming.

If the onset of the fits be definitely periodic, or if a warning be given some time before its actual occurrence, a dose of bromide may often arrest such an attack. Attacks preceded by an aura of a peripheral sensation in a limb may be arrested by constriction of the extremity above the seat of sensation.

The long continuance of bromide administration may lead to a state of bromism, in which the person becomes dull and lethargic, the features become expressionless, the sensibility blunted, the memory and intelligence impaired, the extremities cold; the speech is slow and impaired, and saliva often runs from the mouth. The use of bromide of potassium sometimes leads to an eruption of acne, a condition which may be arrested by a combination of arsenic with bromide, while in other cases cutaneous indurations form, which ulcerate and occasionally attain extensive development of a rupia-like nature.

It is doubtful whether bromide is of equal value in the various forms of epilepsy. Experience shows that it is of less value in the treatment of *le petit mal* than of the severe convulsive forms; and of the latter it is less efficacious in the nocturnal variety. Bromide is partly eliminated through the milk secretion, as bromism has been observed in children suckled by epileptic mothers under treatment by this drug. Bromide exhibited during pregnancy has no deleterious influence upon the fœtus.

To be of value as a curative agent, the administration of bromide should be continued over a long period of time. Even in the most favourable cases, it is not wise to stop its use until two years have elapsed after the last fit; it may then be diminished to a dose once or twice a week, the interval between the doses being gradually prolonged.

Bromide given alone may be the only necessary remedy, but in a large number of cases it may be combined with other drugs. Thus, in the not uncommon cases with weak cardiac action, the combination with tincture of digitalis is of service; while, in the converse cases, aconite or chloral may be for a time judiciously given. In the young and anæmic, bromide should be given along with cod-liver oil, or the hypophosphites and iron.

It is doubtful whether all the bromide salts are equally efficacious. Bromide of potassium appears to be the most valuable; after it come those of sodium and ammonium. A combination of the three is often productive of great benefit, where one alone has not been of much use. Bromide of strontium I have not found equal to the others, although in one case of intractable nocturnal epilepsy it was of undoubted service. Some have found the bromide of nickel a valuable drug.

As bromide fails in some cases, it is necessary to rely upon other, though less efficacious remedies. Perhaps the most valuable is borax, as recommended by Gowers. This may be given in doses of 10 to 20 gr. thrice daily, alone or in combination with bromide. Belladonna, cannabis indica, the zinc salts, nitroglycerin, bromide of camphor, antipyrine, sulphonal, and a host of other remedies, have all been tried, but without any specific result upon the disease. The use of nitrate of silver, a drug largely used before the introduction of the bromides, has been almost entirely discarded.

A method of combining opium and the bromide treatment has been recently recommended by Flechsig, but sufficient evidence has not yet been collected to show that this method is of greater value than that of the bromides alone. A theory of the causation of epilepsy, based upon the presence of excess of uric acid in the blood (Haig), has suggested its treatment by means of salicylate of soda. Many epileptics present errors of refraction; in all, these should be corrected when necessary; but the use of the bromides should not be allowed to fall into abeyance, even though the correction prove of value in lessening the number of the seizures.

Diet.—The diet of epileptics does not require special consideration. The vegetarian régime, advocated by some authorities, has not been found to be of greater value than an ordinary mixed diet. Butcher's meat should not be given at more than one meal per diem; and alcohol should in every case be avoided, unless indicated in emergencies.

W. ALDREN TURNER.

CHOREA.

The name is given to a disease in which the patient is affected with irregular involuntary movements of every part of the body and limbs. The popular equivalent, St. Vitus's dance, has its origin in the history of the epidemics of the Middle Ages, when, in the fourteenth century especially, numbers of people were seized with a hysterical mania for dancing, propagated by imitation, and were only cured by being conducted to the shrine of St. Vitus. But there is really no analogy between the dancing mania, which was also called St. John's dance, and the disease we are here dealing with; and the use of a distinctly medical name for the latter, such as chorea, is desirable. One only difficulty remains, that even under this name some other forms of disordered movement have been described (salaam convulsions, saltatory spasm), which, again, are rather hysterical in their nature. For further distinction, the former, which is very rare, is called *chorea major*, while *chorea minor* is the complete designation of the ordinary chorea; and this whether the symptoms be quite mild, or, on the other hand, very severe, and even fatal.

Etiology.—There are many points of interest in the etiology of chorea, which, in the absence of definite structural changes, are naturally looked to for help in explaining the pathology of the disease.

Age.—Chorea is a disease of youth. Rare in infancy, it occurs especially in childhood and early puberty. Nine-tenths of the cases occur in individuals aged from 5 years to 18 years, inclusive, and two-thirds of these, or about 60 per cent. of the whole, are concentrated on the ages from 9 to 15, inclusive. This increasing disproportion is no doubt in part due to the influence of pregnancy, which balances for a time in females the advantages of a greater age. Exceptionally, chorea occurs in late middle life, or at an advanced age.

Sex.—The female sex is much more liable than the male, in the proportion of nearly three to one. Some statistics have given results which are more nearly five to two; so that the truth may be taken as between these two proportions, or it may be said that the males affected are from 33 to 40 per cent. of the females. Some variations depend on age. Thus, above the age of 15, males are even more rarely affected, unless it be a few in middle or even old age; and the proportion of females to males from 15 to 25 is rather eleven to two than five to two.

Social conditions.—The disease is more common in towns than in the country, and very much more among the poorer classes of society than among the wealthy. Deficient hygienic arrangements have been credited with some share in its production, and it would be difficult at present to deny the operation of such causes, seeing that it is just the poor in our large towns who are especially subjected to them.

Heredity.—The disease is said to occur in so-called nervous families, but the records of ordinary hospital cases do not generally show facts bearing this out. Occasionally it occurs in children of parents who have had it; that is, it appears to be directly transmitted. In other cases, rheumatism has been present in the father or mother, a fact of some interest in connection with the frequent association of rheumatism and chorea in the individual. Hereditary transmission, and the occurrence in several members of a family, are the characteristics of an important group of cases, which also differ from ordinary chorea in the advanced age of the subjects, and the strong tendency to insanity (*Huntington's chorea*).

Antecedent diseases.—The most important of these is articular rheumatism, with which chorea has long been known to have important relations. Numerous groups of cases of chorea have been published, showing the proportion of them in which rheumatism has occurred. The groups are not always strictly comparable, because the criterion of a rheumatic history is not always the same; but the proportion of cases in which a genuine articular rheumatism has occurred previous to the attack of chorea, is probably not less than one-third, and possibly more. In a small proportion of these the chorea occurs in the course of the rheumatic fever itself; in most cases the rheumatism has occurred from a few months to two or three years before. In addition to these, there are some cases in which the patient has an endocardial bruit of unknown causation, presumably, but not proven, rheumatic; and some writers would bring forward as evidence the cases in which the patient's parents have had rheumatism, though the patient herself has not. With these questionable additions, the proportion of cases with rheumatic antecedents would be much increased. But without these the connection must be admitted, unless it can be asserted that articular rheumatism attacks at least one-third of the general population, independent of chorea. I would here remark that the sequence of endocarditis upon chorea, of which I shall have to speak later, does not for one moment prove a connection with rheumatism, since there are other causes of endocarditis besides rheumatism, and chorea may, of course, be such a one.

Anæmia, measles, and other children's disorders, menstrual irregularities, and worms, have sometimes been followed by chorea, but the facts seem at first to help us little as to the nature of chorea, and perhaps to be merely coincidences. Marfan, however, gives statistics of seventy-six cases of chorea observed during four years in the clinique of diseases of children:

and of these, while thirty showed a connection with rheumatism, twenty-eight others had as antecedents some other form of infectious disorder, namely, influenza, five cases; measles, five; scarlet fever, two; varicella, two; chronic bronchitis, with probable tuberculosis, two; boil on the neck, with adenitis, one; dental periostitis, one; impetigo of the scalp, two; otitis media, three; typhoid fever, two; and unnamed suppurative infections, with endocarditis and pericarditis, three cases. The bearing of these facts upon the pathology of the disease will be referred to later.

Pregnancy.—This is a direct exciting cause of chorea in a marked proportion of young women affected with chorea above the age of 17; that is to say, the onset of chorea takes place during the course of the pregnancy, and more often in the earlier months. It is often a first pregnancy, but this may be only because the primipara is more likely to be within the age limits of the disease. Some of them have had rheumatic fever, some have had chorea before, and some have had chorea in a previous pregnancy.

Emotional causes.—The importance of fright as an exciting cause of chorea cannot be overlooked, though it is almost certain that it is over-estimated by the public. The parents of choreic patients are often ready with the most trivial occurrences, which they say have caused a mental shock in the patient, such as a mouse running from under a door, or a man looking suddenly over a wall; and the interval between the event and the onset of the disease is often so long as to make the connection doubtful. But it remains true that numerous cases have developed immediately upon some genuine alarm on the part of the patient, or upon such a mental shock as may follow upon the death of a parent. In the same category may perhaps be placed the cases in which injuries to the head or other part have preceded the disease, though for the present it must remain an open question whether such injuries act directly upon the nervous system or by means of the emotion of fear.

It has long been believed that the disease might be acquired by a child imitating the involuntary movements of another patient; but this is probably a very rare event, and some, if not most, of the cases of allied movements assumed to be acquired by imitation are hysterical rather than choreic in their nature.

Morbid anatomy.—Though the nervous system has been minutely and carefully examined in fatal cases of chorea, no lesions, either macroscopic or microscopic, have been found with sufficient constancy, or of such a nature as to explain the occurrence of this disease. The association of endocarditis with so many cases naturally turns attention to the possibility of embolic processes being the cause. A few observers have found small spots of softening; some others have found embolism of cerebral vessels. But in by far the majority of the cases no such condition is present. Similarly slight changes in the nerve cells, vascular dilatations, distended perivascular sheaths, with leucocytal accumulations therein, and increase of connective tissue—some changes noted in the brain, others in the spinal cord, and others again in the peripheral nerves—do not suffice of themselves to explain chorea, as a large hæmorrhage explains hemiplegia, or suppurative meningitis accounts for coma.

Few of them can be said to be peculiar to chorea; at the same time, some of them may be the indication of other influences upon the nervous system, the common cause, possibly, both of the minute lesions and the functional disturbance. For instance, some of the vascular changes might result from the circulation of a virus or toxine. Others, again, may be the result of circulating disorders occurring within the last few days of life, and brought about by the extreme muscular exertions.

The heart shows much more constant pathological conditions. Almost invariably there is recent endocarditis, even in cases where no murmur has been heard during life. It is in the form of minute granulations, usually on the edge of the mitral valves, occasionally on the aortic valves, or on both. Sometimes there is old or chronic endocarditis. The endocarditis is not confined to cases in which rheumatism is present. In a few cases where chorea has complicated rheumatism, pericarditis may also be present. No other morbid condition is constantly present. In a few instances, micro-organisms have been found in the blood or in the tissues; but they have mostly been streptococci or staphylococci, and nothing of a proven specific nature is yet known.

Pathology.—The pathology of course is obscure, but presents many interesting points. As in most nervous diseases, it is first of all desirable to ascertain the seat of the lesion, and then its nature. The seat of the lesion of chorea is undoubtedly in the brain; this is shown by several considerations—its frequent origin in fright, or other severe emotion; the effect which efforts of the will have in aggravating the movements, and still more the influence which attention has upon them; the fact that the movements cease during sleep; the phenomena of hemichorea, with the important fact that the muscles of the trunk and face on both sides are associated in the movements with the arm and leg of one side only; the implication of the mental faculties to a very slight degree, if at all, in quite mild cases, but seriously in the forms of delusions and maniacal delirium in some cases of *chorea gravis*. When the corpus striatum was thought to be the great centre for muscular movement, the lesions of chorea were sought for in its neighbourhood, but it would be probably in accordance with modern views to look to the cortical motor centres as the seat of the main disturbance of movement in chorea.

As to the nature of the change, the repeated failures to find any constant lesion must reconcile us to the view that it is neither embolism nor thrombosis nor other vascular change, nor inflammation nor degeneration. The last is excluded by the complete recovery which so constantly takes place. And apart from the ill-success of post-mortem examination, the undoubted influence of emotion in its production excludes the possibility of the above anatomical changes being a necessary antecedent. On the other hand, the occurrence of endocarditis in more than half the cases is strongly opposed to the view that it is a purely functional disorder, in the sense that hysteria and neurasthenia are.

The only hypothesis which will satisfactorily associate the functional disturbance of the nerve centres and the very manifest and tangible endocarditis, is the view that they are both the result of some poison or virus or toxine circulating in the body.

Endocarditis is the result of other poisons, such as those of pyæmia and scarlet fever; and there are other convulsive disorders which are not explained by the morbid anatomy of the nervous system, but which are the result of poisons, namely, hydrophobia and tetanus. Moreover, there are some points of close resemblance between hydrophobia, tetanus, and acute fatal chorea. For instance, the involuntary muscular contractions in all three, obviously arising from disordered function of the central nervous system, the short duration of the illness, the rapid exhaustion, the mental symptoms in hydrophobia and acute chorea, and the rapid emaciation in the same two diseases. There is another occasional occurrence which I have noted in all three diseases, which points to the fatal operation of the poison in all three, even apart from the effect of muscular exhaustion, and that is, the entire cessation of the muscular movements for eighteen or twenty-four hours before death. Thus I have seen a man, after suffering from the usual violent movements of hydrophobia, become quite quiet, drink with ease a quantity of fluid nourishment, and yet die within the period common to this disease. A young girl with violent chorea became after a time so quiet that we had hopes that she was recovering, but she died nevertheless within thirty-six or forty-eight hours. And a boy was once sent into the Evelina Hospital whose elbows, buttocks, and scapular regions were deeply ulcerated. The movements at the time of his arrival were so slight that the resident medical officer could not believe the diagnosis. The presence of an apex systolic murmur confirmed the evidence of the history and the sores; the boy took his food well, and had but little choreic movement, but he had some sickness, the temperature rose a little, and he died within six hours of his admission.

The association with rheumatic fever is an additional argument in favour of this view, for those who believe that rheumatism is due to some kind of infective agent. It is not for me to discuss the nature of this last disease; it is sufficient to say that its resemblance to pyæmia and to gonorrhœal synovitis is very close, that endocarditis, pericarditis, and other serous inflammations are only reasonably explained by the existence of a poison. It is true no organism has been conclusively demonstrated in connection with it, but the same may be said of scarlet fever and variola. However, whether the poison is derived from without in connection with microbes, or is derived from within by so-called auto-infection; whether it is a chemical poison, a ptomaine, or a toxine,—the bearing upon chorea is the same

Similar importance must be attached to the fact above stated, that other diseases of undoubtedly infectious nature have been noted as antecedents of chorea; and also to the relation of chorea to pregnancy, for the latter is probably a condition in which infection or auto-infection frequently takes place.

The inclusion of all these separate diseases in the etiology of chorea deprives it of the specific character which its association with rheumatism would lend it, but supports the idea, which has been gradually gaining ground, that it is the result of some kind of infectious process. Marfan regards it as a neurosis provoked by a non-specific infection developing in predisposed soil or tissue, and this predisposition he finds in a "neuropathic inheritance." He states, for instance, that of seventy-six subjects of chorea, forty-nine were the children of neuropathic individuals (epileptic, hysterical, neurasthenic, or psychopathic), and eleven were the offspring of alcoholic persons. These figures, however, require to be confirmed by larger statistics.

A similar explanation is needed for the origin of chorea in fright, which is as much a thorn in the side of a toxic theory as the occurrence of endocarditis is a difficulty in the way of a purely functional disorder. The emotion may act as a depressing agent, and thus determine the operation of the poison upon this part of the nervous system.

In conclusion, it seems probable that, as the convulsive disorders hydrophobia and tetanus are caused by specific viri, and as general convulsions are set up by the toxines of uræmia, by various fever poisons, and by certain drugs, so the movements of chorea are due to toxic agents arising in some way from one or possibly several infectious diseases.

Symptoms.—The onset of chorea is, as a rule, gradual, but in some cases where fright has been a cause the characteristic movements have set in within a few hours. The first thing noticed may be that the child is constantly dropping things, such as cup, spoon, or other article; and then the parents may observe that there is a constant or frequent movement, so that the child appears to be fidgety or restless. Yet in slight degrees or early stages the movements are so slight as to escape the notice of all but practised observers; the finger of one hand is occasionally bent, or the shoulder lifted, or the head turned a little to one side with a quick, sharp movement. In more pronounced degrees many muscles are concerned in every part of the body. The eyes are opened or shut irregularly, the eyeballs are rotated in one or other direction; the angles of the mouth are raised in a smile, or the forehead wrinkled in a frown, unilaterally or bilaterally; the head is jerked to one or other side. If the patient be asked to protrude the tongue, there is often some delay, then the tongue is put out with a jerk, remains in this position for a time, and is then drawn back suddenly as the jaws snap together. The tongue is rarely bitten.

One or more fingers are opened and shut, the wrist flexed or extended, the shoulder raised, and more or less combined movements of flexion, extension, supination, and pronation take place in the arm. In the lower extremities similar movements occur, and the gait is necessarily affected, being slow, halting, irregular, and shuffling; and disorderly, not only from the irregularity of the movement of the legs, but from the want of balancing correspondence on the part of the arms.

In the trunk there are irregular contractions of the thoracic and abdominal muscles and of the diaphragm. The trunk may be twisted round to one or other side. Respiration is gasping or jerky from sudden descent of the diaphragm or from quick contraction of the abdominal muscles.

Speech is irregular and badly sustained, and the patient cannot sing a long note. This is no doubt largely due to the imperfect action of the respiratory muscles. It is nevertheless true that the vocal cords have been seen to quiver, and the patient has a low-pitched, monotonous voice, which is attributed to their deficient tension.

Feeding is often difficult from the irregular action of the tongue and lips, and deglutition may be interfered with in so far as it is a voluntary act.

The condition, so far as the muscles are concerned, is one of—(1) involuntary movement; (2) ataxy or inco-ordination; and (3) a slight degree of actual weakness or paresis.

1. Thus the movements take place during rest, and are quite independent of volition; they are aggravated by voluntary effort, and they are also increased when the attention is drawn to them by the observation or inquiries of others. Except in the most violent cases they cease during sleep.

2. The ataxy is obvious, the patient cannot sit still or walk steadily. The movements are in the highest degree irregular; there is no rhythm at all, no regular alternation of flexion and extension, or any other opposed movements; many of the movements are such as result from the combined actions of two or more muscles, writhing and twisting movements of the

neck, or arm, or trunk. They are rarely movements of great extent even in the more violent cases.

3. The paresis is perhaps not always easy to demonstrate; but the patients are incapable of continued effort in any one direction, and no object can be held for long; the flexors quickly relax, and whether this be due to a primary failing of the impulses, or follow a contraction of the antagonist muscles, it constitutes, as must be recognised, a paresis in the muscles in question. In other instances the patient can scarcely stand or walk, when this defect cannot be attributed to the violence of the ataxy; and in some cases when the involuntary movements have almost ceased, the patients may be quite unable to move arm or leg. Paresis, then, undoubtedly coexists in variable degrees.

As a rule, sensation is unaffected. Definite anæsthesia is very uncommon; only occasionally some pricking, tingling, or numbness, or, on the other hand, hyperæsthesia or even pain, may be present. The reflexes are not materially altered, they may be normal, or slightly diminished, or slightly in excess. The electrical reactions are often difficult to ascertain, especially in young children, but it has been found, at least in some cases of chorea, that there was increased excitability to both faradic and galvanic currents.

Some difficulty is experienced in arriving at a correct estimate of the mental condition of the patient with chorea. Undoubtedly a large number of children with chorea look foolish or even idiotic; but this appearance is in great part accounted for by the misbehaviour of the facial muscles by which the interest and intelligence of the individual are commonly expressed, and which, in this case, contract quite independently of mental processes. But apart from this the child appears dull, and it is easy to see that the emotional condition is altered; the child is fretful, irritable, easily upset, and ready to cry for slight causes, such as the approach of strangers. In the violent cases of older children and young adults, more pronounced mental disturbance occurs, which will be described later.

Of late a good deal of attention has been paid by alienist physicians to the mental condition of choreic patients, and the conclusion of most of these observers is that the mind of the choreic patient is distinctly involved in the greater number, if not in all, cases. On the emotional side there is irritability of temper, capriciousness, impatience, or excitement, on the intellectual side, weakness of memory, absence of mind, and disturbance of the attention.

By one writer this last defect is regarded as the main characteristic, and he refers it to the constant distraction the patient suffers, as a result of the choreic movements. Most writers consider the mental phenomena to be an essential part of the disease. As we shall see later, chorea is a disease of the cerebrum, and the actual cause of it affects at the same time, according to this view, both the motor and the psychic centres.

In the violent cases of older children, and of young adults, and in the chronic incurable cases of middle and advanced age, mental disturbances much more pronounced and readily recognised may occur. They may take different forms in different cases, such as maniacal excitement, melancholic depression, delirium, and hallucinations.

The visceral functions are, with one exception, but little affected. In ordinary cases the temperature is normal, and the gastro-intestinal, renal, and respiratory functions are undisturbed. The exception is in connection with the heart, which, in a notable proportion of cases, presents some abnormality, either clinically or pathologically. In about half the cases of chorea, according to Goodall, a murmur is audible over the cardiac area. This is obviously not confined to cases that have had rheumatism, but a history of rheumatism is much more strongly represented among the cases with murmur than among those without murmur.

The murmur is, in the large majority of cases, heard at the apex and is systolic in time. Of these the majority again are only audible at the apex, or may be temporary and variable; while a smaller number are audible at the base of the chest behind, and may be regarded as certainly due to mitral regurgitation. Many of these last may be associated with such other conditions of the heart, or with such a history, as to make it certain that they have preceded the chorea, and the same will probably be found true of the small number of cases of diastolic or presystolic apex murmur, and of the still smaller number of aortic murmurs. As might be expected in any large collection of young people, largely girls, a basal pulmonary, and probably hæmic, murmur is occasionally heard. What comes out of this is, that in a large proportion of cases of chorea, even independently of rheumatism, a systolic apex murmur becomes audible. The other conditions of the heart will vary with the antecedents. In the obviously long-standing cases of valvular disease, some enlargement or displacement of the apex, or irregular action may be expected. In most cases, however, the apex is in its normal position.

But even in cases where it may be supposed that the murmur is secondary to the chorea, and the size of the heart is normal, some irregular action is often noticed. This is readily explained by the action of the respiratory muscles; as it is impossible that the diaphragm, intercostals, and abdominal muscles should contract with the characteristic jerk and irregular way without influencing the beat of the heart.

As to the cause of the cardiac murmur, there can be little doubt that it is due to a true endocarditis, even in those cases where there is no history of rheumatic fever. The evidence lies in the fact that pronounced valvular disease may develop afterwards in such a case, and still more conclusively in the fact, that at the post-mortem inspections of the comparatively rare cases which are fatal, endocarditis, either of the aortic or mitral valves, is almost invariably found. In many of these cases, but not in all, a murmur has been heard during life.

There is thus little necessity to discuss the theory, that the murmur is due to irregular choreic contraction of the papillary muscles controlling the closure of the mitral cusps.

Varieties.—Like other diseases, chorea may be mild or severe: and there is no broad line of distinction to be drawn between the different grades which may be observed. In the very mild cases, only one or two muscles may be moved, whereas, in the pronounced, yet moderate cases, many parts or every part of the body may be involved.

In a small proportion of the cases, the limbs of one side are affected, while those of the other are still. This is called hemichorea: it happens in the milder form of case, and the right and left sides are affected with about equal frequency. But it is interesting to note that though the chorea attacks the limbs on one side only, the muscles of the trunk and face are affected on both sides. In some cases, the chorea disappears from the side first affected, and then appears in the limbs of the opposite side.

In another case, the movements are so violent in every part of the body, that life is threatened, and in a small proportion of cases is lost. The movements are constant and more extensive than in ordinary cases. The patient is unable to stand, and, even if in bed, must be confined by the clothes or bed-bands to prevent her falling out. Feeding is difficult, as the head is not still for a moment. The constant friction of the limbs and under surface of the trunk against the bed rapidly induces abrasion and ulceration of the skin, so as to cause sores of a circular or oval form on the heels, buttocks, and elbows, and over the scapulæ. Occasionally delirium or a maniacal condition is superadded, and the patient may die, from what appears to be exhaustion, or from septic poisoning. These cases (*chorea gravis*) are much more frequent in young adults than in children. They are not necessarily violent from the first; they may begin as mild cases, and after some days or weeks, develop the more serious conditions. Another variety may perhaps deserve a name, namely, that in which the paresis of the limbs is very pronounced; this has been called paralytic chorea.

The rare cases in which people in middle or advanced age acquire chorea, are often spoken of as varieties, adult or senile chorea. The movements are materially of the same nature, and the course and duration may be no different, that is, the patient may recover in a few months; but more commonly the disease is incurable, and persists for several years, or until death. Among these adult cases, with a persistence of the chorea, there is an increasing liability to the supervention of definite insanity, which, moreover, is likely to be of a depressed or melancholic type, perhaps progressing to stupidity. And this mental complication is almost certain to occur in those adult or middle-aged cases referred to, in which there is a hereditary or family history of the same disease (*Huntington's chorea*).

Diagnosis.—The diagnosis of chorea presents few difficulties; it is usually at once recognised from the nature of the movements. In some cases of hysteria, movements may occur which are very like those of chorea, but they are more rhythmical, more simple, and less combined than in chorea; more limited to one or other limb or part of a limb; more constantly and uniformly repeated. The condition is sometimes called hysterical chorea; but as it is really hysteria and not chorea, I believe it would be wiser to speak of it as choreic hysteria.

The jactitating and jerky movements of Friedreich's disease might give rise to some confusion with one who had seen but little of this disease; but the gait is quite different; the history is one of long duration; nystagmus is present, and the movements are rather jerky, ataxic modifications of voluntary movements than quite involuntary movements, like those of chorea. An analogous distinction applies to the wide but irregular tremors of disseminated sclerosis, which occur, moreover, only on attempts at movement.

Prognosis.—The duration of ordinary cases of chorea is from one to three months. The recovery is always gradual, and there is, so to say, a slow conversion of a serious case into a mild one, and then the involuntary movements finally cease altogether. The rare exceptions to the rule, that chorea is of a few weeks' duration, are the cases occurring in adults, to which allusion has been made.

It is, however, common for relapses to take place, and patients may have the disease two, three, or four times, or even more often. The later attacks may, one or more, be induced by the same cause as brought on the first attack, or the apparent causes may be different, as for instance, fright in a first attack, and pregnancy in a second.

The fatal cases have usually a short duration, which will depend rather upon the time at which the symptoms become aggravated, or upon other complicating conditions. In uncomplicated cases the death is usually attributed to the exhaustion from the violent movements. The patient takes but little food from the mechanical difficulty of administering it, emaciation takes place with startling rapidity, the surface of the skin is reddened from friction, and abraded or ulcerated as above stated, and the secretions are necessarily passed in the bed. Yet death does not always occur in the midst of violent movements; on the other hand, the movements may have entirely ceased, or may consist of a mere occasional twitching of a finger, for twenty-four hours or more before death takes place.

In other cases, there are complicating conditions of active, acute rheumatism, or valvular disease, or pericarditis, which no doubt contribute to the fatal result. And in others, the wounds caused by friction may set up septic infection.

Treatment.—The first thing to be done in a case of chorea is to keep the patient quiet, to remove the child from school, and let it rest in bed. In the more pronounced cases this is absolutely necessary, and it can do nothing but good even in the quite mild varieties. Every source of irritation should be avoided, and the child should be protected from the ridicule of companions, and spared the worry of study. The food should be abundant, and easily digestible; milk, bread and butter, farinaceous puddings, and eggs may be given, with fish or meat once in the day.

If there is any obvious and removable cause, it should be dealt with. Coincident rheumatism should be treated, worms should be removed, even pregnancy in a very severe case might require to be terminated; but, as a rule, there is little to be done in this direction, and one turns to drugs for what help can be got from them.

The movements of chorea have been regarded as indicating a weakness on the part of the nervous system, and accordingly the drugs called nervine tonics have long been in request. Iron and zinc have been employed largely; the former as tincture of the chloride, the latter mostly as the sulphate; and it used to be the practice to give increasing doses of zinc sulphate, beginning with 2 gr. three times a day, and increasing the dose by an additional grain (three times a day) every two or three days. More recently the value of arsenic has been very widely admitted, and it does appear sometimes rather rapidly to diminish the activity of the movements, when they have persisted with little improvement for some time. A dose of 3 minims of liquor arsenicalis, or of the liquor sodii arseniatis, may be given three times a day, and this should be increased every two or three days by the addition of 1 or 2 minims to each dose, until the child may be taking 10 or 15 minims three times in the day. Some patients are so susceptible to the influence of arsenic, that these larger doses are not reached, but the medicine has to be omitted for longer or shorter intervals and then resumed. Still, even in the case of arsenic, one must recognise that the symptoms generally disappear rather slowly. Other drugs which have been used are the bromides, in doses of 5 or 7 gr. to a child of 10, hydrobromic acid in $\frac{1}{2}$ drm. doses, antipyrine in doses of 5 gr., three times a day. Of these the last seems more often to have given satisfactory results. Strychnine has also been given, and seems to be more especially useful in cases where the paresis is marked. In children that are anæmic or badly nourished, iron and cod-liver oil may be taken in addition.

I have seen good from daily massage in rather pronounced cases; and in quite mild cases and in recovering cases, various kinds of gymnastic exercises have been used. If the more modern views of its pathology are correct, these methods can only act as adjuvants, and can do little towards dealing with the *fons et origo mali.*

The violent and severe cases present the greatest difficulties. To feed the patient, to save her from injuring herself, to prevent exhaustion and to procure sleep, are tasks which will indeed tax the resources of the physician. The food must be fluid and concentrated, and must be administered in teaspoonfuls constantly; for instance, milk, eggs, beef-tea, beef-jelly, or beef-essences. The patient must be in some way restrained and protected from the chance of injury. The sores, however, come not so much from blows as from friction; the prominent parts of the back are ulcerated, and abrasions occur even on the knees from contact of the overlying clothes.

The bed should be provided with padded boards, or, perhaps better, the limbs may be carefully bandaged with flannel, and so bound, the legs together and the hands to the side, a blanket being placed across the body and hips to steady them as much as possible (Broadbent).

Medicinally, one is tempted to give the more powerful sedatives, such as chloral, chloroform, or morphine, and the difficulty is to steer an even course between giving too little to get an effect, and giving too much for the good of the patient. The administration of chloroform will afford temporary relief, but the continuous administration sometimes attempted is not satisfactory. Chloral may be given by the mouth or by the rectum, morphine by subcutaneous injections, and small doses rather frequently are preferable to large single doses. The effects in any case must be very carefully watched; as it is, it may be by no means easy in a fatal case to be sure how far the final symptoms have been contributed to by the medication employed.

FREDERICK TAYLOR.

CHOREA IN ADULTS.

It has been already stated that exceptionally chorea occurs in adults of middle age or advanced years; and this is often termed adult or senile chorea.

Some of these cases present no essential difference from the chorea of childhood. The symptoms may develop at almost any age, and in both sexes, and they may be preceded by emotional or mental causes, but they are not specially related to rheumatic fever, nor does endocarditis result from them. The movements are very similar to those of the disease in younger cases. The duration is variable; it may be a few months, or one or two years, but much more often the condition persists to the end of life. The results of pathological inquiry are not much more satisfactory than in juvenile chorea; certain degenerative changes may be found, but they are probably secondary.

The other group of adult cases is that already more than once mentioned, in which the disease has a strong family or hereditary tendency. These cases were first described by Huntington of Pomeroy, Ohio, and hence it is known as Huntington's chorea, or hereditary chorea. It has occurred in the individuals of four generations, and in several members of a family, or in more than one family descended from the original victim. It commonly begins between the ages of 35 and 50, and persists until death. The movements begin in the face and hands, and eventually all parts of the body may be involved. Sensation and the reflexes are generally normal, but the mind gradually gets impaired, and the condition goes on to a pronounced dementia.

Degenerative changes have been found in the brain, but here also it is possible and probable that they are secondary. Opinions differ as to whether this should be considered as a variety of ordinary chorea or as an entirely distinct disease. A wider acquaintance with these hereditary cases, and more certainty as to the nature of juvenile chorea, are necessary before either opinion can be confirmed.

FREDERICK TAYLOR.

PARALYSIS AGITANS.

Syn., Shaking Palsy; Parkinson's Disease.

This disease, originally described by Parkinson in 1817, is a progressive disease, commencing in late middle life, characterised by muscular tremor, weakness, and rigidity, associated in the later stages with a peculiar gait, and a characteristic attitude of the head and neck. The tremor, however, is not essential for the diagnosis of this condition; cases are occasionally seen in which rigidity and weakness are the prominent phenomena, more especially in the face and hands. These cases have received the appellation of *paralysis sine agitatione.*

Etiology.—As a rule the disease commences in the sixth decade of life, to a less degree between 40 and 50 and between 60 and 70 years of age. It is rare under 30, but a few instances have been recorded. Of these the disease is said to have commenced at 17 years of age (Berger), and as early as 12 and 16 (Charcot).

Heredity probably has little influence in the causation of this disease. Gowers and Berger place the number of cases in which this has been noted at about 15 per cent.; in fact, in many instances longevity in one or both parents is a remarkable feature.

Of exciting causes little is known; some ascribe the commencement of their disease to cold, others to mental anxiety and shock; while in others it seems to be directly the result of traumatism. It has been pointed out by Gowers that physical injury may determine the limb first affected. Of two cases of fall on the shoulder, the tremor commenced in the arm on the injured side. Charcot mentions a case of contusion of the thigh, which was followed by tremor in that limb; and a case of dislocation of the jaw, followed by tremor in it; in both instances the tremor persisted and became general. Specific conditions, such as syphilis, acute infectious disease, and toxic agents, have less influence in the causation of paralysis agitans than in most nervous diseases.

Pathology.—Various pathological conditions have from time to time been described as constituting the morbid anatomy of this disease: but in the majority of cases these are non-essential, and are met with in other conditions than paralysis agitans. Of such may be mentioned degenerative changes in the smaller blood vessels of the central nervous system, and increase of the neuroglial tissue. These changes outside the nerve elements proper, with pigmentation or vacuolation of nerve cells, are conditions seen in other degenerative diseases, and as a result of senile change.

That the actual cause of the tremor is to be found in alterations in the cells of the cerebral cortex, is probable, although no definite observations have been obtained on this point. The resemblance which this disease has in some respects to the traumatic neuroses, and the fact that the tremor has been known to come on suddenly, both as a result of injury and of fright, would seem to suggest a functional or nutritive basis for the onset of the symptoms. Hence we are inclined to regard the varieties of the disease as of this nature; in the one set—those of sudden origin—the shock may determine a nutritive alteration, which may eventually become permanent; while in the more common form, of gradual onset, the protoplasmic change goes *pari passu* with other indications of a degenerative nature.

Symptoms.—The symptoms of this disease are considered under the following headings, which form its chief characteristics:—

Tremor.—This commences usually in the limbs, chiefly in the upper limb on one side, and almost invariably in the index-finger and thumb. Cases are met with, however, in which it begins in the shoulder. The tremor is rhythmic, characterised by alternating opposition and extension of the thumb, with flexion and extension of the fore and other fingers—productive of the well-known "cigarette-rolling" or "bread-crumbling" movement—pronation and supination of the wrist. These movements may be seen, from a slight but characteristic tremor up to marked shaking of the whole limb. In the leg the tremors are usually about the ankle-joint; and more especially in the calf muscles, causing a condition resembling ankle-clonus. Movements of the lower jaw are also noticed more rarely, as well as flexor and extensor movements of the head. Rotation of the head is rare. Gowers has met with a case of tremor in the orbicularis palpebrarum, and Westphal in the lower facial muscles. Tremor of the tongue is also rare. Tremor of the vocal cords and respiratory muscles is only exceptionally observed.

The feature by which the tremors of this disease may be distinguished from other forms, is its continuance during repose, and its suspension during the execution of voluntary movements. It is present whatever position the patient occupies, be he sitting, standing, or lying down. It may be temporarily arrested in an effort to stop it, but shortly returns with increased vigour and enlarged range. An attempt to arrest the tremor in one arm may cause it to temporarily overflow to the other. It is not usually increased in ordinary voluntary movements; but cases have been mentioned by Gowers, in which all the other features of paralysis agitans were present, and in which a coarse tremor was started, on volitional effort. The movements are usually quiet during sleep and anæsthesia.

The onset of the tremor is, as a rule, slow, usually beginning in the thumb and forefinger, but occasionally in other parts, as already stated. The so-called hemiplegic type is the commonest form of invasion, in which first the arm and then the leg on the same side become involved. A decussated form has been described, in which the arm on one side and the leg on the other are implicated, but this is rare. More usually does the tremor affect, first one arm and then the other, and later on the legs.

Muscular rigidity.—Muscular rigidity is as important an element in the disease as tremor. In some cases it is the earliest and only phenomenon. Its existence is productive of several of the characteristic features of the disease. It more especially involves the muscles of the neck, back, and face, but largely also those of the extremities. Hence the face assumes a peculiar and pathognomonic expressionless appearance; it is like a mask, the person neither smiling, laughing, nor frowning. The attitude also becomes changed from the normal; the head is bent forward, as well as the upper part of the spinal column, which assumes a curved posture in the upper dorsal region. The arms are carried in front, the elbows slightly away from the chest, the forearms flexed, and the hands flexed at the metacarpo-phalangeal joints, and extended at the others. The attitude of the hand closely resembles that seen in chronic rheumatoid arthritis, but there are no enlargements about the ends of the bones. The legs are slightly flexed, and there is usually adduction of the thighs; and if the rigidity be excessive, forms of talipes may be developed in the feet, and sometimes a claw-like contraction of the toes.

Hence the patient's gait becomes peculiar. He rises cautiously and with difficulty; hesitates for a short time, and then starts, at first slowly and with increasing rapidity; as has been said, he appears to be running after his centre of gravity. To this feature has been given the term festination or propulsion. In rare instances a condition of retropulsion has been noted, in which there is a tendency to run and fall backwards. All cases of paralysis agitans, however, do not show these special features. Although propulsion may be a leading feature of a case, retropulsion need not necessarily be also present.

Associated with the rigidity, is a weakness or difficulty in carrying out active movements. The muscular actions are slow, the grasps feeble: an interval elapsing before the muscles respond to the will. So also are all those movements of a more complex nature, such as dressing and undressing, rising from a chair, or turning round suddenly. There is no true defect of articulation, nor any slurring speech; but the voice is monotonous and destitute of modulation. Often there is difficulty in starting speech, but, once commenced, the words follow each other quickly.

Rarely does the weakness ever amount to complete paralysis; even in the last stages a certain amount of movement is retained. The knee-jerks are usually unaffected; a false ankle-clonus, arising from tremor, may be started when the foot is dorsally flexed. The muscles retain their normal volume and electrical irritability.

Sensory symptoms.—Although the characteristic features of paralysis agitans lie in the domain of motion, there are certain subjective sensory phenomena occasionally described. The one of which patients most commonly complain is a sense of heat, either local or general, but it does not appear to be associated with any marked rise of temperature. Owing to this sensation, these sufferers are more comfortable in cold than in hot rooms, and dislike much clothing. Increase of perspiration is often associated with it. Much rarer is a subjective sensation of cold. So-called "rheumatic" pains about the joints of the limbs are often complained of in the early stages; while a feeling of unnatural fatigue is not at all uncommon. Objective sensory phenomena do not occur.

Course.—The disease is chronic and progressive. It begins locally, and tends to become universal. Local tremor, usually in the hand, may or may not be the first symptom; the mask-like condition of the face being often a very early feature of the disease. On the other hand, in some cases an acute beginning has been described. The tremor may remain for some years limited to the limb in which it commenced, but almost invariably spreads to the others.

The disease is of long duration, and appears to have little tendency to cause death, except in so far as by its debilitating action it favours respiratory and other complications. It has been known to last from fifteen to thirty years, but the average duration is from ten or twelve years.

Diagnosis and prognosis.—The diagnosis is easy in a well-marked case; but less from the nature of the tremor than from the facial expression, the general attitude, and the gait. It is, however, necessary to distinguish the tremor of paralysis agitans from that of disseminated selcrosis, and from ordinary senile tremor.

The following table shows the chief characteristics of these three conditions:—

Paralysis Agitans.	Disseminated Sclerosis.	Senile Tremor.
1. Tremor continuous during rest.	1. Tremor only on volitional efforts (so-called "intentional tremor").	1. Tremor more on voluntary movement than during rest.
2. May be arrested temporarily by voluntary effort.	2. Quiet during repose.	2.
3. Rhythmical, and usually fine.	3. Coarse and irregular.	3. Rhythmical and fine.
4. Affects one limb or limbs more than the others.	4. Observed chiefly in the upper limbs.	4. Chiefly and early affects the head and limbs on both sides.
5. Rarely affects the head.	5. In well-marked cases, head set in motion with tremor of the arms.	5. A condition met with in extreme old age.
6. Associated rigidity. Characteristic facies and gait; a disease of late middle life.	6. Often nystagmus; sometimes slurring utterance and unsteadiness of gait; a disease of youth.	6. Absence of rigidity.

The prognosis is largely influenced by the history of the origin of the disease, being more favourable in the cases of acute onset. The disease has little tendency to shorten life, but the chance of amelioration is at the best slight.

Treatment.—This is a disease for which little can be done of a permanent nature, although temporary improvement is not uncommon under appropriate treatment. Drugs are of little service; nerve sedatives, such as the bromides, chloral, hyoscine being of as little value, as strychnine, which usually tends to increase the muscular rigidity characteristic of the disease. Cannabis indica combined with arsenic has been found by Gowers to be most efficacious.

Most reliance should, however, be placed upon the use of galvanism, in conjunction with well-regulated and carefully carried out passive movements of the affected limbs. Cases are not uncommon in which this method of treatment has been of marked temporary benefit. Passive movement of the joints of the limbs to a large extent mitigates the rigidity, which is almost as distressing a feature of the disease as the characteristic tremor.

W. ALDREN TURNER.

SPASMODIC TORTICOLLIS.

Torticollis or wryneck is a term which may be applied to any distortion of the neck of the nature of lateral rotation. It is not, as a fact, generally used for the purely temporary condition attributed to muscular rheumatism, and more commonly known as stiff neck, in which the extension of the muscles of one side of the neck is painful, and ease is sought by inclining the head to the affected side; nor to such want of symmetry from muscular contraction on one side as results from the irritation of the nerves by caries of the cervical vertebræ. But it is more strictly reserved for two forms of permanent lesion, one of which is fixed or congenital wryneck, the other is spasmodic wryneck. In the former, the head is drawn down to one shoulder by the permanent shortening by structural change of one sterno-mastoid muscle; and this change is with little doubt, in some cases, the result of a partial rupture with hæmorrhage at the time of birth. As the child grows, considerable asymmetry occurs. If the lesion is on the right side, the head is always too much to the right, the middle line of the face falls to the right of the middle line of the sternum, the right side of the face is smaller than the left, the right orbit somewhat lower than the left, the right angle of the jaw more square than the left, and the middle line of the face appears to be curved with the concavity towards the right.

Spasmodic wryneck, or spasmodic torticollis, on the other hand, is an acquired disease, and is characterised by involuntary tonic or clonic contractions of the sterno-mastoid muscles and of other cervical muscles, by which the head is twisted into abnormal positions and generally to one side.

Etiology.—The disease is certainly rare, and the cause is by no means always apparent. It occurs in early adult and middle life, and it is more frequent in women than in men. Neurotic inheritance is traceable in a good proportion of the cases. Other instances have been attributed to cold or overwork, either mental or physical; and physical work, involving much use of the muscles of the arm, neck, or shoulder, has been observed in some cases.

Pathology.—This is quite obscure. No anatomical lesion is known as a course of spasmodic wryneck, and the rarity of the disease in comparison with the frequency of occurrence of the events to which it is occasionally attributed, make it difficult to understand the connection between them. The long persistence and chronic course of the disease, and the practically perfect health of those who suffer from it, prevent one attempting to find an explanation in toxic or infective agents. One is reduced to the conception of a functional excitement of some part of the neuro-muscular apparatus, and it is even open to doubt which particular portion of that apparatus is really the seat of the lesion assumed. The analogy of other disorders in which clonic spasms of muscles takes place, renders it practically certain that the lesion is neither in muscle nor in nerve fibre, but in nerve cells: but whether it is chiefly the cortical or the spinal cells whose functional activity is morbidly stimulated, is not at all easy to decide. Possibly the one may be more affected in some cases, the other in others. Thus, in some the movements are more those produced by muscles in combined action, in the others the muscles are acting simply or independently. Experiments have shown that excitation of the cortex cerebri causes tonic and clonic convulsions, while stimulation of the lower centres causes only tonic spasm.

Symptoms.—The onset is gradual, it occurs as it were spontaneously, and for no obvious reason, in the midst of perfect or practical health. Some discomfort is felt in the neck, and sooner or later it is realised that the head is being turned aside in a way not desired. The sensations or movements may be but slight at first, or only occasionally, as for instance when the patient tries to write; but after a time the movements become well marked and more or less constant.

The movements are of course effected by contractions of the muscles, and these contractions are sometimes slow, continuous, or tonic; at others, they are quick, jerky, and alternating with relaxations, that is, clonic. In different cases the tonic or the clonic variety may predominate, at different times in the same case one or other may be in excess. Cases have been described as being tonic, or clonic, or tonico-clonic; probably the great majority of the cases are of the last combined kind.

Assuming, for purposes of illustration, that we have a case in which the right sterno-mastoid muscle is alone involved, the movements are of the following kind:—The head is turned slowly to the left with the chin slightly raised, the occiput depressed; the patient, by an effort of the will, and with the help of the antagonistic muscles, forces the head back into the median position. Directly the effort is relaxed, the right sterno-mastoid again forces the head round; then there may be a sudden jerk, and the chin is carried further; or a succession of short jerks, and the chin is carried right round over the left shoulder. The spasm relaxes then for a time, and the head goes back to its median position; or its backward movement is again interrupted by the insistence of the muscle concerned. In mild cases the movement is but slight, and is easily controlled, or, if yielded to, itself soon relaxes; but practically in most developed cases the movements are incessant, and continue all day; while in severe cases the movements are quite violent, the tonic contractions are conducted with great force, and the clonic movements are violent starts and jerks, which are painful in themselves and accompanied by facial contractions expressive of the discomfort or distress they occasion. All these contractions are aggravated by attempted movements of the head or limbs, and even by walking or moving; and they render difficult, not only eating and drinking, but also various other everyday acts. They are increased also by any attention directed to them by friends or observers, in the same way as are the movements of chorea, athetosis, and some other tremulous and spasmodic disorders.

The movements in developed cases continue all day, and scarcely leave the patient a minute's peace during his waking hours; they may be so severe as to prevent his sleeping without sedatives; and if he sleeps the contractions begin again directly he wakes, so that he scarcely recognises that they have ceased during sleep.

What has been said of the sterno-mastoid applies to the other muscles of the neck, which are so often associated with it in the convulsive disorder. It is probable that all the muscles having attachments to the occipital and mastoid portions of the skull are in some or other cases involved. Thus, most commonly, the trapezius is affected with spasm; but the spasm is confined to the upper part, and the middle or lower portions are free. The splenius is also frequently concerned, and also the deep extensors and rotators of the head, namely, the recti capitis postici major and minor, the obliquus superior, and obliquus inferior, and the rectus lateralis.

The complexus and mastoid may also assist; and the scaleni are also said to be affected with spasm, though their influence on the movement of the head can only be subsidiary by their action in flexing the neck. Of these muscles, nearly every one, by drawing the occiput or mastoid back towards the middle line, causes rotation of the head, that is of the face, towards its own side, and accordingly will operate for this common object with the sterno-mastoid of the opposite side. The complexus acts slightly in the contrary sense, that is, it turns the face a little to the opposite side, but its main action is directly backwards.

In different cases the muscles concerned are variously grouped. The sterno-mastoid muscle of one side is most commonly associated in its action with the cervical muscles of the opposite side, so that the combined result is a decided rotation of the head (or face) away from the side of the spastic sterno-mastoid. In other cases the cervical muscles of both sides may act together with the sterno-mastoid of one side. In others again the cervical muscles of one side and both sterno-mastoids may be in action together, one sterno-mastoid probably much more than the other. Sometimes the sterno-mastoids are but slightly affected, and the posterior cervical muscles of both sides are chiefly concerned, by which the head is constantly pulled backwards, and comparatively little rotation takes place. In these cases there is often an associated action of the occipito-frontalis muscles, and the eyebrows are involuntarily raised as the head is pulled back. In a minority of cases, other muscles besides those of the head and neck are the seat of spasm; for instance those of the upper arm, of the face, and of the jaw. In a case that I observed several years ago, not only some spasm of the arm was associated with that of the sterno-mastoid and posterior cervical muscles, but the lumbar muscles on one side were constantly the seat of clonic contractions of precisely the same nature.

Diagnosis and prognosis.—This is not, as a rule, difficult. The constant movements, whether of a slow, forcing, or quick, jerky character, distinguish spasmodic wryneck from other cervical distortions, which are mostly fixed. Such, for instance, are the strained position and limited movement of stiff neck; the similar condition resulting from caries of the cervical vertebræ; the rotation of the head in conjugate deviation, in which the displacement of the head is generally accompanied by deviation of the eyes to the same side; and the fixed torticollis already alluded to, in which the sterno-mastoid is permanently shortened by changes occurring at or about the time of birth. The only real difficulty is in reference to hysteria. The fact of the occurrence of these spasms in a woman, together with the fact that the movements are aggravated by close attention, may lead one who is little familiar with them to suppose that they are hysterical, or even wilfully assumed. Prolonged and constant observation of the patient will generally dispel these suspicions. On the other hand, there may occasionally occur in hysteria muscular contractions taking on this type: but they are likely to happen to much younger females than are the sufferers from spasmodic torticollis, and to be associated with other hysterical manifestations.

The course of the disease is variable, but for the most part it is unfavourable. Some cases have got well after a longer or shorter time. The majority are little amenable to treatment, and the symptoms persist with varying degrees of improvement, or with none at all, for many years or even the remainder of life.

Treatment.—The few remarks on prognosis have indicated that treatment is not by any means satisfactory. Our complete ignorance of its pathology leaves us no rational basis to go upon, and we have to depend almost entirely upon symptomatic or empirical methods. These may be grouped under the following heads:—Nervine tonics, sedatives, galvanism, mechanical restraint, and surgery. Arsenic, iron, zinc, asafœtida may be included in the first group. Gowers records some success from a combination of asafœtida and valerianate of zinc; but the others do little if any good. By means of sedatives, there is no doubt that the movements can be controlled; but the rest continues only during the few hours the influence of the drug persists, and as the drug is eliminated the spasms return. Chloroform, opium, and morphine, by subcutaneous injection, are the more powerful remedies of this class. I have seen good from the use of full doses of bromide of potassium, and the spasms, which, it is true, were not very severe nor of long duration, subsided almost completely. If opium or morphine is extensively used in a severe case, there is, of course, the fear that the morphine habit may be induced. Large doses, such as 2 or 3 oz. of succus conii, have been given with temporary success, the muscle being thus paralysed; but when the conium is discontinued the spasms recur. If galvanism is tried, it must be by means of a weak constant current, applied continuously during five or seven minutes to each muscle. Mechanical apparatus can only be applied in quite mild cases. Patients often antagonise the spasmodic movements, by the contraction of the opposite muscles, or by the use of the hands, or by fixing the head on the pillow, or against the head of an arm-chair, or otherwise, so as to prevent the extreme displacements effected by the morbidly contracting muscles. Such patients may be materially assisted by metal, felt, or plaster apparatus, taking its bearings from the shoulders, and fixing the head as nearly as possible in the normal position. This method makes, of course, no attempt to cure the disease; it can only relieve the symptoms partially; and in cases with severe movements the apparatus cannot possibly be borne.

Finally, we may call in the assistance of surgery. Naturally, one of the first things done was to divide the spinal accessory nerve which supplied the sterno-mastoid muscle. But the spasm returns as the nerve unites; and even if a portion of the nerve is excised so as to prevent this, there are objections in that the muscle is largely paralysed and atrophied as a result, that the trapezius is paralysed and atrophied as well, even in its lower portion, which does not join in the spasm; and, lastly, that the spasm must still persist in such other cervical muscles as were simultaneously affected. The nerve has been also stretched and ligatured. Of recent years the principle has been more thoroughly carried out by the division of the posterior branches of the cervical nerves, by which these other muscles are supplied. Good results have been thus obtained, but it can only be by the substitution of a tolerable for an intolerable inability. Muscles must be paralysed and atrophied, but the patient is happy while free from the constant twitching and spasm, even if the movements of his head, neck, and shoulder are not what they might be. It has generally been thought that division of the muscles themselves was little likely to be of service, and that the portion left in connection with the nerves would contract, and cause pain and discomfort, although not productive of torsion effects upon the head. Kocher of Berne has, however, treated several patients by the systematic and repeated division of the muscles, and satisfactory results are claimed for this method in a good proportion of the cases, namely, cure in seven cases out of twelve, and improvement in three others. Kocher divides the sterno-mastoid about the level of the angle of the jaw, the trapezius at the level of the mastoid process, the splenius, the complexus, the trachelo-mastoid, and the obliquus inferior. On which side they are divided depends on the variety of the spasm. Several operations may be necessary, because when the muscles cicatrise the movements may return. Why, in a certain number of cases, or after a second or third division, the movements do not return, has not been clearly explained. Even in cases that are practically cured a certain amount of discomfort is experienced, consisting of tension and pressure in the muscles of the neck, brought on or aggravated by fatigue or emotion, and sometimes amounting to pain.

One patient, treated in successive operations by division of the left sterno-mastoid and right cervical muscles, and by removal of a portion of the left sterno-mastoid, was unable to write with the right hand, because the attempt was soon followed by rotation of the head to the right side.

FREDERICK TAYLOR.

OCCUPATION NEUROSES.

By this term is meant a condition of cramp, tremor, or paresis of some muscles engaged in the carrying out of certain professional movements, such as writing or pianoforte-playing; the cramp thus produced causing inability to carry out the actions in question. In the early stage of these conditions, other movements of the same muscles are sometimes possible, though in the later periods all movements of the affected muscles are impaired. Various forms of occupation-cramp have been described, some of which are etiologically pure neuroses, arising from fatigue of the centres in the spinal cord; while others are associated with the onset of chronic nervous disease, or with local affections of the joints or muscles chiefly concerned. Hence, in discussing the causation of these conditions, there have to be considered—(*a*) the influence of fatigue of the nerve centres, apart from the presence of organic disease; (*b*) the association of fatigue with degenerative conditions, as hemiplegia, progressive muscular atrophy, paralysis agitans, and amyotrophic lateral sclerosis; (*c*) the existence of local arthritic affections—tenosynovitis, and gouty deposits. The true cases of professional neuroses naturally fall into the first class above described.

Pathology.—In these, the muscular cramp arises from nutritive impairment of certain cell-groups in the anterior horns of the spinal cord, a condition which may eventually pass into temporary paralysis in severe cases. This condition is more prone to arise in those centres which have been specially educated for a particular action, and are, in consequence, more prone to fatigue; or in those which, regulating certain ordinary movements, have been worked to excess. But the incidence of organic nervous disease, as already stated, may be productive of phenomena similar to those arising from fatigue alone. Thus writer's palsy may be the earliest indication of a commencing hemiplegia; in a case of clarionette-player's palsy, the special movements of the tongue required to play this instrument were the only signs of a hemiplegia, which had in other respects cleared away.

Symptoms.—The commonest occupation neurosis is seen in the so-called writer's cramp, or palsy. But whether we are studying a case of this condition, or of the closely-allied tailor's and pianist's cramp, or of the more general muscular weakness illustrated in the palsy of treadlers, hammermen, or sawyers, certain general nervous symptoms are found associated with the local spasmodic or paralytic features. These are—

1. Tenderness along the course of one or more of the nerves of the affected limb, or tender points over certain nerve branches. This may in some cases arise from peripheral neuritis, but in others is merely of a neuralgic nature
2. Diminished faradic excitability of the muscles whose movement is impaired. More rarely, and only in severe cases, has the reaction of degeneration been demonstrated.
3. Tremor of the affected limb.
4. The onset of early fatigue and a sense of discomfort or restlessness of the affected limb chiefly at night.

It has been pointed out by Poore, to whose researches I am chiefly indebted for an account of the several trade neuroses here described, that the spasm affects those muscles which are subject to prolonged strain. Hence, in writer's cramp, the muscles concerned in "pen-prehension" rather than those of "pen-movement" are involved; in pianoforte-player's cramp, the muscles which maintain the wrist and fingers in an extended position, rather than those which flex the fingers; in sawyer's cramp, the muscles which fix the humerus at the shoulder-joint, rather than those effecting a to-and-fro movement of the arm; and, in hammerman's cramp, the muscles which fix the scapula to the chest wall are those whose movements are impaired.

Before proceeding to a consideration of the several forms of professional neuroses, it may be stated that this term is employed to distinguish them from those affections of a toxic nature, such as lead palsy, mercurial tremor, etc., which, though trade diseases, are due to obvious organic disease.

Writer's cramp.—A person suffering from this condition finds, shortly after starting the act of writing, that the pen is not under proper control; the strokes may be too long, or too short; he presses unnaturally upon the paper; a painful state of the muscles of the hand ensues; spasm of the abductor indicis occurs, and the pen drops from between the forefinger and thumb. Further attempts at writing merely augment the discomfort.

The muscles chiefly involved in this act are the first and second dorsal interossei; the opponens, abductor, and flexor brevis pollicis, and the extensor primi internodii pollicis—these being muscles of pen-prehension. In severe cases, or those in which treatment has not been adopted, other muscles become involved—forearm, upper arm and shoulder muscles.

In the early stages, it is probable that other movements of the affected muscles may be effectually carried out; but it is usually found that the muscles implicated in one action perform other actions in an imperfect manner. Writer's cramp is a disease chiefly of the male sex.

Pianoforte-player's cramp.—In this condition cramp affects the muscles which maintain the wrist and proximal phalanges in an extended position, which is the attitude of the limb enjoined by the "Stuttgart method." By this method the muscles most subjected to prolonged strain are the extensor communis digitorum, the dorsal interossei, and the extensores indicis, and minimi digiti. The local condition of which complaint is made, is that the fingers tend to "run together" and are unable to be lifted from off the keyboard. The complaint may affect both arms, but more commonly only one, and that the left. It occurs chiefly in young women of delicate health who are in the habit of "practising" for many consecutive hours. Other movements, involving the use of the forearm muscles, are usually implicated at the same time. The nerve tenderness in these cases is chiefly over the musculo-spiral nerve and its branches.

Needle-plier's cramp.—The tailor holds his needle in much the same way as the writer his pen; that is to say, the muscles involved in needle-prehension are similar to those of pen-prehension, but with the addition of the flexors of the phalanges of the thumb and index-finger. In this condition, which is very rare, the thumb and forefinger give way, so that the needle is no longer grasped with sufficient firmness. The tenderness is chiefly of the median nerve, but the ulnar may also be involved.

Hammerman's cramp.—In the cases of this condition which have been recorded, there has been found a more general affection of the movements of the arm, than in those forms of occupation-cramp already described. Some of the cases have been hemiplegic in character, while others were of the nature of a brachial monoplegia. The serratus magnus muscle, which fixes the scapula, would appear to be that chiefly subjected to prolonged strain in this action. Spasm of all the arm muscles is brought on by attempts at the action of hammering, so that the aim of the right arm, which holds the hammer, becomes imperfect. Cerebral symptoms would appear to be commoner in this condition than in the others; an explanation for which is offered by the fact that co-ordinated movements of both arms and the eyes are necessary in the taking aim, which is required of the hammerman.

Sawyer's cramp.—On attempting to use the saw, disorderly movements of the arm occur, preventing its proper use. The muscles subjected to strain in this condition have been found to be the supraspinatus and the pectoralis major, chiefly its clavicular portion; these being muscles which fix the head of the humerus in the glenoid cavity.

Cornet-player's cramp.—I have described a case of cornet-player's cramp, in which the movements of the tongue necessary to produce staccato notes were defective. In this case all other tongue movements were readily performed. In another case, a clarionette-player, who had in other respects recovered from an attack of hemiplegia, was unable to effect movements of the tongue necessary to play this instrument.

A number of other forms of occupation neuroses have been described—telegraphist's cramp, turner's cramp, cigarette-maker's cramp; also treadler's, compositor's, gold-beater's, dancer's, and many others.

Diagnosis and prognosis.—The recognition of a true case of occupation-cramp is rarely difficult; the onset of the symptoms with the special act, be they pain or spasm, is not easily mistaken. As already indicated, a difficulty in a special professional action may be indicative of an early stage of a chronic and progressive nervous disorder: such as disseminated sclerosis, tabes dorsalis, hemiplegia of gradual onset, peripheral neuritis or paralysis agitans.

The prognosis of such cases is variable. If the patient comes under treatment in the early stages, and the occupation is given up entirely for a time, a favourable result may ensue. If, however, other muscles than those chiefly concerned in the professional ailment be affected, or movements other than those chiefly required for the special action be involved, then the outlook is grave. Persistence at the occupation under difficulties, for weeks or months after the onset of the first symptoms, may lead to an incurable condition. But yet cases are observed of an apparently hopeless nature, which recover under rest and judicious local treatment.

Treatment.—In almost all cases of occupation neurosis, the patient is in a poor state of general health. The value of general and nervine tonics should therefore not be overlooked. For the local condition, of the first importance is rest from the occupation which has given rise to it. This is absolutely necessary for the treatment to be of any service; for the sooner, after the onset, the patient is able to stop work, the more favourable is the chance of recovery. Combined with rest in the early stages, local counter-irritation over the tender nerve trunks, or tender spots, is of value in allaying pain and discomfort.

Of the two forms of electricity in common use, galvanism is that which is of most service in this condition; although a combined galvano-faradic application is recommended by some physicians. Faradism by itself is of little value and may be harmful in cases with much spasm. Galvanism may be materially assisted by judicious kneading and massage of the muscles of the affected limb; but this should only be carried out after the local nerve tenderness has been allayed by rest and counter-irritation.

W. ALDREN TURNER.

COMA.

By this term is meant a state characterised by loss or defect of consciousness, usually associated with stertorous breathing and a tendency to death by asphyxia. Of the several forms of defect of consciousness, coma is the most intense and the most serious. Of less severity are profound sleep, from which the individual may be awakened; lethargy, from which he may be momentarily aroused; and stupor, a condition in which, with apparent obliviousness of surroundings, the person may be aware of passing events, and the reflexes are not lost.

Etiology.—The natural termination of many acute nervous conditions is in coma. Thus an epileptic convulsive fit invariably ends in this way; thereby denoting temporary, complete, and universal paralysis of the higher centres, as a result of a previous excessive discharge of energy. In cerebral hæmorrhage of ingravescent character, all the stages of coma, from slight blunting of intelligence up to complete loss of consciousness, may be observed in a single case. In many forms of insanity, and more especially in general paralysis of the insane, the picture of a gradually invading coma is striking. In alcoholic intoxication also, it is not difficult to study a similar sequence of events, well expressed in the words of Mercier: "Consciousness, so far as we can judge of it by its outward manifestations, marches steadily from the light, through a penumbra of greater or less breadth, into the darkness of oblivion. As with the mental strength, so with the bodily strength. There is a continuous loss of muscular power, ending in complete powerlessness. The slight unsteadiness becomes a reel, the reel ends in a fall, the struggles subside, and the patient lies like a log, the picture of utter prostration."

Symptoms.—In coma there is paralysis of the voluntary, and partial paralysis of the involuntary muscular system, the respiratory and cardiac centres being least affected. In death from coma, the respiration fails before the heart, the interval sometimes extending to several minutes. Stertorous breathing is a measure of the degree of unconsciousness, as it indicates a complete abolition of reflex action with falling back of the tongue, and paralysis of the soft palate. Stertor may be arrested by pulling forward the tongue, or laying the patient upon his side.

In general medicine, however, the conditions which give rise to a state of coma have to be more particularly studied. Hence it is important to consider whether there are any symptoms or signs, by aid of which coma, arising from this or that cause, may be determined. In every case of coma the practitioner should obtain a sample of the urine (by catheterisation), and test for the existence, or otherwise, of albumin and sugar; take the temperature of the body; note the state of the respiration, pulse, pupils, and the knee-jerks; examine the skull; and when possible endeavour to obtain information as to the nature of the onset.

Diagnosis.—When presented with a case of coma, several possible causes should be borne in mind.

Alcoholic intoxication.—Unless an obvious history of drinking be obtained, it is almost impossible to distinguish the unconsciousness of an alcoholic bout, from that of cerebral hæmorrhage, or fracture of the base of the skull. An alcoholic odour of breath, or the presence of alcohol in the urine,[1] is not a sufficiently clear signs, for liquor may have been given after the onset of the symptoms. The following positive and negative evidence taken together is strongly in favour of alcoholic poisoning: a marked alcoholic odour in the breath, the presence of alcohol in the stomach-washings and in the urine, unassociated with the signs of fracture of the skull (*e.g.* bleeding from the ears, nose, or mouth), of albuminuria and glycosuria, and of unilateral paralysis. In all cases of this nature coming to the casualty department of a hospital, the following question has to be answered: Is the patient drunk, or is he drunk and dying from some associated condition, such as cerebral hæmorrhage or fracture of the skull. In mild cases, the question may usually be answered after a short interval, especially if the stomach has been washed out; but in many no decision can be given at the outset. Hence in all cases of doubt the patient should be given the benefit and treated for the severer malady.

Uræmia.—As already pointed out, the presence of albuminuria in a comatose person does not necessarily imply uræmic coma. In this variety, the coma is usually less profound than in that due to cerebral hæmorrhage. The age of the individual is the point on which most reliance should be placed, with a view to diagnosis. The presence of albuminuric retinitis would confirm the existence of uræmia. Uræmia is common in the young, rarer in the old. The presence of pus in the urine would be favourable to the diagnosis of a pyæmic condition arising from pyelo-nephritis.

Narcotic poisoning.—The onset of this form of coma is usually slow; the pupils are markedly contracted, the respirations infrequent, and the skin moist. In rare cases a sudden onset of coma from opium poisoning has been described.

Coma may terminate some cases of diabetes mellitus, but the presence of large quantities of sugar in the urine and the characteristic odour of the breath associated with acetonæmia are usually sufficient to establish the diagnosis of this disease; but not always, for cranial injury may occasionally give rise to glycosuria.

An epileptic fit.—The coma following an epileptic seizure is usually of short duration, the patient partially recovering consciousness, though in a dazed state, in from a few minutes to an hour. On recovery there is no

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[1] If a drop or two of alcohol-laden urine be put into the following mixture, a bright emerald green coloration is produced—

Pot. bichromat.	1 part.
Ac. sulph. fort.	300 parts.

paralysis, and the tongue may have been bitten. The knee-jerks are usually exaggerated, and an ankle-clonus may be obtained. Should a history of convulsion immediately preceding the onset of coma be obtained, the probability is that the condition is of epileptic nature; but it should be borne in mind that some forms of cerebral hæmorrhage—the so-called epileptiform apoplexy—begin in this way; as also may the coma of uræmia and of general paralysis of the insane.

Cerebral hæmorrhage.—It is not usually possible during the stage of coma to determine into which portion of the brain hæmorrhage may have taken place. In pontine hæmorrhage the pupils are stated to be contracted to pin points, but there are cases on record in which they have been found dilated. A large hæmorrhage into the lateral ventricle or basal ganglia does not usually give rise to any localising symptoms, although rigidity of the limbs on the side opposite the lesion has been observed. In less profound states of coma, some amount of unilateral paralysis may be discerned; and the knee-jerk may not be obtained on the side of paralysis. Coma may also follow upon a subdural hæmorrhagic effusion, the onset of which may be heralded by convulsions. In apoplectic coma the temperature is usually raised after an initial depression; a steady rise of temperature with deepening coma indicates a fatal termination. In the coma of pontine hæmorrhage, hyperpyrexia is stated to occur; but this is also observed towards the end in hæmorrhage into other parts of the brain, more especially the basal ganglia. The presence of albuminuria in coma occurring in an elderly person is highly suggestive of cerebral hæmorrhage.

Fracture of the skull.—The coma associated with this condition is almost invariably due to the presence of cerebral hæmorrhage from laceration of the brain, either the direct result of the blow or from *contre-coup.* The history of a blow or fall is commonly obtained; in most cases there is external evidence of injury, such as a cutaneous wound, or subcutaneous ecchymosis, or bleeding from the ears, nose, and mouth. In these cases the hæmorrhagic effusion is not necessarily limited to the membranes or surface of the brain. I have made post-mortem examinations on several cases, in which were found hæmorrhagic foci in the basal ganglia unassociated with degenerative disease of the arteries.

Organic brain disease.—Several forms of organic disease of the brain, other than hæmorrhage, may manifest themselves in convulsions followed by coma. It is not usually possible during the comatose state to differentiate these forms from each other or from apoplexy. The conditions to which reference is made are: general paralysis of the insane, intracranial tumour, and abscess. The points to which attention should be paid are: the age of the patient, the existence or otherwise of optic neuritis, the absence of albuminuria, and the presence, or a history, of ear discharge.

Sunstroke.—Insolation is very rarely seen in this country, but the climatic conditions prevailing at the time, the history of the case, and the pyrexia, are usually sufficiently characteristic.

Patients are sometimes brought to the casualty department in a state of stupor, the cause of which is difficult to determine. Of etiological factors, catalepsy and cerebral syphilis should chiefly be borne in mind. It is not improbable also that some cases of this nature are purely hysterical.

W. ALDREN TURNER.

GRAVES' DISEASE.

Syn., Parry's Disease; Basedow's Disease; Exophthalmic goitre; Struma Exophthalmica; Cardiothyroid Exophthalmos; Tachycardia Strumosa Exophthalmica.

THIS is a disease characterised by protrusion of the eyes, enlargement of the thyroid gland, and frequent action of the heart or tachycardia. With these three cardinal symptoms are usually associated muscular tremor, mental excitability, vascular dilatation, and increased metabolism, with concomitant symptoms.

Etiology.—Sex has a marked influence upon the number of cases affected. The proportion of men to women varies so much in the statistics of different authors that it is hard to give it exactly, but it may be roughly stated to be at least six or eight times more frequent in women than in men. It has been observed in children at the age of 2½, and also as late as 68, but it is commonest in women from 15 to 30 and in men from 30 to 45. The patients are often of delicate build and nervous temperament, and generally belong to neurotic families, in other members of which hysteria, epilepsy, diabetes, and mental diseases have appeared. Dieulafoy has observed cases in three successive generations, and a case is recorded of an hysterical woman who had ten children, eight of whom and also three grandchildren suffered from this disease. I myself have seen it in two sisters, and have observed it in the case of a girl whose mother was suffering from myxœdema. Climatic or telluric conditions probably influence it. Lebert states that it is commoner in Breslau than in Switzerland or France, but Eichhorst says that it is much more common in Zurich than in the towns of North Germany. This author also noticed that it appeared to him more common in schoolmasters than others. Amongst the exciting causes, mental shock, grief, and fright are the most common; but over-strain, mental or bodily, and depressing diseases, such as influenza, appear to predispose to it.

Morbid anatomy.—Increased vascularity of the medulla, slight hæmorrhages in the floor of the fourth ventricle, as well as hyperæmia, swelling, atrophy of the nervous elements, or sclerosis of the ganglia in the cervical sympathetic chain, have been described. Hale White has pointed out that the recurrent laryngeal nerves appear to have a trophic influence on the thyroid gland, and Cyon has found that the branches which pass from the laryngeal nerves to the thyroid contain vaso-dilating fibres which, when irritated, greatly increase the stream of blood through the gland. The circulation of the thyroid is also greatly increased by stimulation of the depressor nerves, so that great exertion, emotion, or other influences acting on the heart, may, through the depressor nerves, increase the thyroid circulation. These observations may help to explain the effect of emotion in producing thyroid hypertrophy, while many of the symptoms, as already described, may be produced by the increased secretion of the gland consequent on such hypertrophy. Nevertheless, the pathology of exophthalmic goitre cannot be regarded at present as at all certain. According to Cyon, the iodothyrin formed by the gland greatly increases the excitability of both the depressors and the vagi. From its action on the vagi it ought to slow the heart, but the effect upon the depressors would be to dilate the blood vessels and quicken the heart, while iodine and the iodides have an opposite action.

Pathology.—The pathology of this disease is not yet definitely settled. It probably is due in part to some lesion of the cerebral nervous system, and in part to auto-intoxication by the thyroid secretion. Thyroid extract, when given in excess, produces tachycardia, tremor, sweating, and great subjective feelings of heat, while exophthalmos has been reported as due to an overdose. In exophthalmic goitre the thyroid gland undergoes true hypertrophy of exactly the same kind as occurs after partial excision. It seems probable that in this disease the secretion from the hypertrophied gland is increased in quantity and acts as a poison to the organism, but that the disease does not depend wholly upon such auto-intoxication. In some cases the symptoms are well marked, while the hypertrophy of the gland is comparatively small. It may be urged that in such instances a part of the gland below the sternum may have undergone hypertrophy, but it has not been shared to the same extent by the part of the gland which has been exposed. Nevertheless, proof is wanting of this contention. Moreover, the hypertrophy of the gland appears to be preceded in many cases by tachycardia and mental disturbance, and no explanation has yet been given of why the gland should hypertrophy. The most probable explanation of the disease is, that it owes its origin to a central nervous lesion, leading to disturbed innervation of the heart, and probably also to enlargement of the thyroid gland, which in its turn produces auto-intoxication. By injuring the restiform bodies, Filehne succeeded in producing tachycardia, exophthalmos, and enlargement of the thyroid, though he did not succeed in producing all three symptoms in one animal. The rapid action of the heart, staring eyes, and moist skin are commonly associated with the emotion of fear, and this emotion is frequently felt in an exaggerated form, and for insufficient causes, by patients suffering from this disease. The seat of the emotions has not been localised, but when assisting my friend Dr. Ferrier in some of his experiments upon the localisation of function in the brain, I watched the results while he applied the electrodes to the parts, and I was struck with the fact that, in galvanising the corpora quadrigemina, emotional excitement seemed to be produced in some of the animals. It appears to me not improbable that the origin of the disease may yet be found in the corpora quadrigemina, but this is hypothetical.

Symptoms and course.—In a well-marked case the eyes are prominent and staring, the throat is thick, so that the patient is obliged to wear a larger collar, the pulse is frequent and soft, the force of the cardiac beats is frequently increased, the skin is moist and warm, and the hands tremble. The patient complains of feeling nervous and irritable, of being easily excited and frightened, of disliking crowds or even of seeing strangers, of palpitation, and frequently of shortness of breath, and generally of feeling uncomfortably hot even in cold weather.

On examining these symptoms more particularly, we find that the eyes are not only more prominent, but the opening between the eyelids is larger than usual, the upper lid being retracted so as to show the sclerotic above the iris, and the eyes have an unwinking stare (Stellwag's sign). On asking the patient to look at the finger, held straight before the eyes, it is found that, on lowering it, the upper lid does not follow the movements of the eyes, so that the strip of sclerotic between the iris and upper eyelid becomes wider, if it already exists, and becomes apparent in the slighter cases where it was not previously noticeable (von Graefe's sign). There is sometimes weakness of the upper eyelid, producing ptosis, and generally there is weakness of one or more of the muscles of the eye, such as the internal recti, causing strain or imperfect convergence (Möbius' sign). Occasionally the external recti are not only weak but paralysed, giving rise to diplopia, and sometimes ophthalmoplegia occurs. The amount of exophthalmos varies much in different persons, and it may vary in the same patient at different times. It not infrequently affects one eye rather more than the other, while the lobe of the thyroid is usually though not always larger on the side corresponding to the greater protrusion of the eyeball. The eyes frequently water, but are sometimes abnormally dry, and on account of the absence of winking and the imperfect closure of the eyelids the cornea may undergo ulceration. The protrusion of the eyeballs is sometimes very great, so that the eyelids do not close even in sleep, and occasionally, though rarely, the eyeball may be quite uncovered by the eyelids. Complete dislocation of the globe has been recorded. Vision is unaffected, as a rule, although subjective sensations of light may be observed. The pupil is usually normal. Sometimes a certain amount of œdematous swelling under the eyelids has been observed. On ophthalmoscopic examination, the veins of the retina have been found enlarged and tortuous, the disc somewhat congested, and pulsation has been seen in the retinal arteries.

The enlargement of the thyroid is usually symmetrical, but not infrequently is greater on one side than the other. On palpation it not infrequently pulsates visibly, but it is not always easy to determine how much pulsation is communicated from the carotid, and how much is actually due to pulsatile enlargement of the gland itself. On palpation, it feels soft and elastic in most cases, but there may be adenomatous nodules present in it, giving rise to a feeling of hardness or nodulation. An expansile pulsation and often a thrill may be felt on palpation. On auscultation, a loud, more or less continuous, humming murmur is generally to be heard, increased at each systole, but sometimes the humming may be almost or completely absent and the systolic murmur alone heard. I have also heard a very loud, soft, superficial systolic murmur over the manubrium sterni, and for about an inch or rather more to each side of it in a case of great enlargement of the thyroid. This, I think, was probably due to extension of the gland into the anterior mediastinum.

The pulse rate is very frequent, varying from 90 to 180 or more. It is readily excited by emotion or movement. It is commonly regular, but may be irregular or intermittent. The cardiac beat is generally forcible and is frequently much diffused, and may be so powerful as to be evident even through the dress. The apex-beat and cardiac dulness may both be normal, but if the disease is of long standing, dilatation or hypertrophy, or both, generally occur, so that the apex becomes displaced outwards and the cardiac dulness increased. An anæmic murmur over the pulmonary artery is very common, and a systolic murmur at the apex by no means infrequent. There is often also a loud murmur over the carotid arteries, and a venous hum may be heard in the neck. There is general dilatation of the arteries, the carotids throb much, and there is very generally a systolic murmur over them. The radial pulse is frequent and soft. In some cases it may be full and large, but it is often small and soft, on account of the frequency of the heart's action and the consequent smallness of the wave of blood sent into it at every beat. The capillaries are also well filled, so that the skin is generally warm and moist. A capillary pulse may usually be seen, and a venous pulse may sometimes be observed in the veins of the hands.

Tremor of the hands is another symptom of almost equal importance with the three already described. The tremors are small and frequent. They are usually about 8 or 10 per second, or about double the rate of senile tremor or paralysis agitans. I have, however, seen the rate fall to four vibrations a second. The tremor occurs also in the feet, and may extend to the whole body. Subjective feelings of heat are very common. The temperature as a rule remains normal, but in all probability this is due to the great loss of heat from the skin, which is both warm, from the free circulation of blood in it, and moist. Much heat is thus lost both by radiation and evaporation, so that the temperature is usually prevented from rising, in spite of the heat generated by increased tissue change. The increased metabolism in this disease tends to cause great emaciation.

The electric resistance of the skin is markedly diminished. This is probably due to the free circulation and moisture already mentioned. Muscular weakness is frequent, patients getting easily tired; and occasionally the legs give way suddenly, causing the patient to fall. The tendon reflexes are generally rather exaggerated than otherwise. Mental changes are well marked in most cases, though not in all. The patient generally becomes nervous and irritable, easily frightened and easily worried. Sleep is often light, restless, and easily broken. Associated with these symptoms one may find the emotional and moral alterations frequently observed in hysteria, so that the patient may alternate between great depression and exaltation, agreeable manners and extreme perversity; sometimes melancholia or mania occurs. When the latter is acute, it is a bad omen. Cutaneous eruptions not infrequently occur—pigmentation, leucoderma, urticaria, and pruritus. The nails sometimes become thin and corrugated. Localised swelling is sometimes observed, and is probably of a neuro-angiotic character, not to be confounded with anæmia or cardiac dilatation which may occur in these patients.

The condition of the tongue varies much; it is generally fairly clean. The appetite may be either poor or excessive, some patients being always hungry. The digestion may be good, but the patients are liable to acute attacks of diarrhœa without any apparent reason, and sometimes this may be associated with violent vomiting. The breathing is usually frequent, sometimes 30 or 40 per minute, and, like the pulse, it is quickened by exertion or emotion, and nervous cough may occur. The expansion of the chest may be greatly diminished. Albuminuria is not infrequent, but this is probably sometimes due to cardiac dilatation, consecutive to the disease. Diabetes is not uncommon. Menstruation is usually little affected.

The course of the disease varies much. The first symptom usually appears to be an alteration in the temper, often noticed by the patient's friends, or a feeling of nervousness felt by the patient herself. This may lead to a medical man being consulted, and the frequency of the pulse being then discovered. Sometimes, however, palpitation may be the first symptom to attract the patient's attention. In other cases the first change observed is protrusion of the eyes, noticed by the patient's friends, or she may first find that she requires a larger collar. The symptoms usually come on slowly and successively, in the course of some months, but occasionally they may come on quite suddenly. In one case which I have recorded, the patient was greatly agitated by a thunderstorm, and in thirteen hours or less the eyes had become protuberant. In this case the disease became chronic. Solbrig reports a case where the disease became fully marked in about a day, but disappeared again in ten days; while Osler quotes a case in which the symptoms came on with great rapidity, and the patient died on the third day. Usually the disease comes on slowly, runs a chronic course, and gradually improves. The effect of marriage on exophthalmic goitre is uncertain. In some cases, where the marriage is sterile, the course of the disease is accelerated and the symptoms aggravated, but in others the occurrence of pregnancy is distinctly beneficial or even has a curative effect.

Diagnosis.—When all the three cardinal symptoms, namely, goitre, exophthalmos, and tachycardia are present, the diagnosis is certain, but sometimes one or other symptom may be absent, and in such atypical or *fruste* cases there may be some difficulty, and then other minor symptoms must be taken into consideration. The condition most likely to be mistaken for it is simple goitre occurring in anæmic and nervous persons. But simple goitre is generally more firm and less elastic, and although it has been said to have caused exophthalmos and quickened pulse by pressure on the sympathetic, yet the protrusion of the eye will be one-sided and probably accompanied by dilatation of the pupil, whilst in exophthalmic goitre affection of the pupil is rare. The pulse also is likely to be only slightly quickened when the patient is quiet. But in such cases the diagnosis rests chiefly on the absence of the following symptoms, namely, v. Graefe's, Stellwag's, and Möbius' signs—pulsation of the retinal arteries, tremor of the hands, mental and emotional excitability, warmth and moisture of the skin, subjective feelings of heat, and diminished electrical resistance.

Prognosis.—This is uncertain, but a considerable number recover. The proportion of these is difficult to ascertain, as most of them remain unrecorded. So far as my own experience goes, most of the cases improve or recover, but their course is slow, and they remain under treatment for about three years, after which they disappear from observation, either because they are quite well or so far recovered that they think further advice unnecessary. A certain amount of swelling of the thyroid and prominence of the eyeballs may remain. In some, the condition continues with slight variations for a good many years. In very severe cases with rapid emaciation, or in those complicated by diabetes or cardiac disease, the prognosis is unfavourable. The patients rarely die of the disease itself, unless a profuse watery diarrhœa, which nothing will stop, be reckoned as part of it. Usually they are carried off by some intercurrent febrile condition, such as pneumonia. Statistics appear to show that death occurs in about 25 per cent. of the cases. It may occur from marasmus, due to increased metabolism, or from persistent vomiting, choleraic diarrhœa, or cardiac failure. The latter may be preceded by dyspnœa, possibly due in some cases to enlargement of the thyroid below the sternum, but in others it may occur without any premonitory symptom whatever. In other cases death may occur, with symptoms of gradual cardiac failure and mitral incompetence. In most cases death is due to intercurrent disease, especially of a febrile character, such as pneumonia or bronchitis, the heart, which in ordinary conditions is beating as fast or faster than is usual in high fever, being quite unable to meet the demands of the system when the temperature is high. Sometimes death may occur with no warning, and so quietly as to be unnoticed until some time afterwards, as in a case which I described.

Treatment.—The most important factor in treatment, probably, is to give the patient as much rest, bodily and mental, as possible. If the case is severe, she ought to be confined to bed and kept quiet at least for two or three months, and any news or information likely to cause excitement should be withheld. In less severe cases, gentle exercise in the open air, short of fatigue, is advisable, but should on no account be carried so far as to produce exhaustion or strain, nor should the patient be allowed to do anything or see anything likely to cause excitement. The diet should consist of an ordinary plain mixed diet of farinaceous food, fish, chicken, and a moderate proportion of butcher's meat, vegetables, and fruit. If any craving is experienced for unsuitable or highly spiced articles of diet, the patient should not be allowed to take them, but the craving will probably be satisfied by placing a pinch of salt on the tongue and allowing it to dissolve. If this is insufficient, some solution of citrate of potash may be slowly sipped. Tea and coffee, which are powerful cardiac stimulants, should be avoided in very severe, and only used in moderation in less severe cases. Alcohol as a rule had best be abstained from, and tobacco should be used sparingly if at all. Change of air and scene are often useful, and a long sea voyage supplies these as well as very complete rest. Residence at some of the Swiss Alpine resorts is sometimes beneficial. In severe cases, cold applied over the thyroid gland, by means of Leiter's tubes, appears to be beneficial, and wet packs and massage, by equalising the circulation, are also to be recommended, unless they are found to excite or worry the patient.

Amongst medicines the substances most likely to antagonise the effects of excessive thyroid secretion is suprarenal tissue, which has an action upon the circulation almost exactly opposite to that of the thyroid gland. I have tried this in a good many cases, and it has appeared to me to have very considerably ameliorated the symptoms, although I cannot say it has produced a complete cure. Extract of thyroid gland, as one might expect, generally makes the patient worse. Extract of thymus or compressed thymus has been stated by some to be beneficial. I think I have also found benefit resulting from chloride of calcium, in 5-gr. doses three times a day, continued over many months. Amongst the drugs especially used to quiet the heart's action are digitalis and strophanthus. These sometimes are useful, but not so much so as one would expect. Nerve sedatives are, on the whole, most satisfactory, especially belladonna and bromide of potassium. Belladonna may be given in 5 to 10 minims of the tincture (B.P. 1898), and the bromide may be given in 10, 20, or 30-gr. doses, three times a day. If this is found to cause too much depression, some aromatic spirits of ammonia may be added to it, and if there is any tendency for the temperature to rise, especially at night, 15 or 20 gr. of salicylate of soda may be combined with it. Amongst medicines which may be regarded as special foods are iron where anæmia is present, arsenic as a hæmatinic, and nervine tonics such as hypophosphite, and glycerophosphate of soda. Phosphate of soda, in doses of ½ drm. to ½ oz. daily, has been very highly recommended. Cod-liver oil, petroleum emulsion, or malt extract may be given as general nutrients, and if the bowels are at all constipated, extract of cascara may be mixed with the malt extract. If vomiting occurs, it is probably best checked by 3 to 8 minims of dilute hydrocyanic acid, given with 20 gr. of bicarbonate of potash and 17 of citric acid in effervescence. If it is not readily checked, nutritive enemata should be employed to maintain the strength of the patient. The diarrhœa is exceedingly difficult to treat, and all remedies may fail to arrest it. Bismuth, opium, catechu, logwood, and other astringents may be given by the mouth, and the bowel washed by large enemata, to which the remedies just mentioned, or sulphate of zinc, half a grain to the ounce, may be added.

Galvanism has been strongly recommended in this disease, but I have been personally disappointed with its results. A weak current should be passed from the forehead and eyes to the occiput, from one mastoid to another, and from the back of the neck to each side of the trachea, so as to influence both the thyroid and the sympathetic. A weak current may also be passed from the epigastrium to the back of the neck, and from the left inframammary region to the lumbar. The direction of the current may be reversed from time to time. De Watteville mentions a case where a strong current, applied by the subaural method with a large electrode over the lower cervical and upper dorsal vertebræ, and a smaller one under each ear, gave good and rapid results. Other authors recommend faradisation instead of galvanism.

Of late, surgical treatment has been employed with comparative frequency. It is somewhat difficult to judge of its results, inasmuch as the cases have differed, not only in the operations used, but also in the severity of the cases. In considering the results of operations for exophthalmic goitre, the exceedingly good results of simple enucleation of adenomata ought to be excluded. The operations employed have been—(*a*) ligature of the thyroid arteries, (*b*) division of the isthmus, or (*c*) removal of one lobe or a portion of the lobe. Removal of the whole thyroid, of course, tends to produce either death or myxœdema, and is therefore not employed. The results of operation may be seen from the subjoined table. One may say that, roughly, over 40 per cent. are put down as either cured or much improved. This may not seem a very much larger proportion of recoveries than the number already mentioned as occurring without operation; but when it is considered that in most cases where the operation has been undertaken the usual treatment has probably been first of all tried and found inefficacious, it seems probable that operation may occasionally be useful. One would not be inclined to try it, however, unless other remedies had all failed.

Result of Operations on the Thyroid Gland.

	Collected by	
	F. Kinnicutt.	M. Allen Starr.
Cured	60	74
Improved	71	45
Unaltered	11	3
Died	13	23
Result unknown or doubtful	25	45
	187	190

LAUDER BRUNTON.

RAYNAUD'S DISEASE.

Syn., Symmetrical Gangrene.

This term denotes a somewhat variable group of paroxysmal vasomotor disturbances, originally described by the author whose name it bears, in which three different phases may be recognised—local syncope, local asphyxia, and local gangrene, all of which have a marked tendency to symmetry.

Etiology.—The affection is undoubtedly more common in women than in men, but perhaps not in the great disproportion indicated by Raynaud. It may begin at any age, but many cases seem predisposed to it from birth; in some instances it is hereditary. A significant feature in this connection is the generally recognised fact that many cases have been preceded by malaria. Cardiac disease is occasionally associated with the affection; this, however, is probably accidental. The combination of Raynaud's disease with hysteria and neurasthenia is frequently observed.

Among the causes which excite a paroxysm must be mentioned cold; the attacks are not only more frequent in winter, but they are distinctly precipitated by exposure to low temperature. Many nervous disturbances, such as shock, anxiety, or apprehension, are also responsible for the onset of the paroxysms.

Morbid anatomy and pathology.—The post-mortem examination of patients who have died after showing the manifestations of the affection is sometimes absolutely negative; there may, nevertheless, be evidences of loss of tissue with cicatricial results, or even the appearances of recent gangrene. The most noteworthy lesion hitherto described is neuritis, more especially of the extremities chiefly affected, as in the cases described by Affleck and Rakhmaninoff. This lesion is far from constant, and may safely be held to be, as Judson Bury suggests, an epiphenomenon. Many more observations are required before the exact relations of such peripheral nerve lesions to the symptoms can be estimated.

The only acceptable view in regard to the pathology of Raynaud's disease refers the condition to a vasomotor neurosis, specially implicating the circulation of the extremities. It is extremely probable that future investigations may lend support to the suggestion advanced by Bury, that there may be two or three pathological varieties of the affection: the principal lesion in one type being in vasomotor centres; in another type, implicating vasomotor nerves; while, perhaps, in a third group the change may affect the whole vasomotor tract, involving cells as well as fibres.

The appearances of local syncope may be explained by a simple spasm of the vessels; the asphyxia probably requires to be explained by a dilatation of the capillaries and veins before the arterial spasm has passed off; the gangrene, originally held by Raynaud to be the result of a previous asphyxia, may undoubtedly take its origin directly from the ischæmia of local syncope, as well as from a hyperæmia following upon asphyxia.

Symptoms and diagnosis.—In reviewing the symptoms of Raynaud's disease, it is necessary to consider the three phases or grades of intensity already mentioned, as well as certain associated phenomena.

Local syncope.—This phase closely resembles the effects of extreme cold, and it results in the production of a state very like that known as dead fingers. As a rule, when the patient is seen, this phase is found along with the next grade of severity, some fingers being as white as snow, while others are of a deep crimson or even purple tint. Although the fingers and toes are those most frequently affected, the ears also suffer not infrequently, while the nose and parts of the arms and legs may be implicated.

Local asphyxia.—This stage is usually a sequel to local syncope, but it may occur primarily. The parts affected become swollen and hyperæmic, so that the fingers may be absolutely livid. While the syncope is attended by numbness, this stage is usually accompanied by a sensation of stiffness and soreness, and there may be pain of an agonising character. On the other hand, there is often total anæsthesia and analgesia, objective as well as subjective.

Local gangrene.—Many degrees of death of the tissues may be sequels of the preceding stages. Most commonly, in chronic cases, small patches of necrosis are found at the tips of the fingers, toes, and ears. These may begin, as already indicated, at the stage of syncope, although they more commonly show themselves after the phase of asphyxia. The terminal portions of one or more of the fingers or toes assume a black appearance, and are found to be cold and anæsthetic. Vesicles appear near the extremity and the skin necroses until a line of demarcation is established, and the affected portions slough away. This process may involve a very large loss of tissue.

Associated phenomena.—Several interesting symptoms are associated with the local manifestations of the disease. The digestive system is, as a rule, sluggish, and if it be overtaxed, there is an increased tendency to attacks, while, after paroxysms have occurred, jaundice has been observed. This possibly results, as Abercrombie suggests, from a vasomotor spasm of the hepatic vessels. The blood undergoes considerable alterations. The corpuscles are crenated, and do not form rouleaux as usual, while granules make their appearance. Even when the blood in the general circulation is normal in character, such features may be seen locally in the affected regions. One of the most important symptoms is hæmoglobinuria, which has been frequently observed. The usual sequence of events is that the hæmoglobin appears two or three hours after the onset of the local syncope. Blood corpuscles are rarely present, but oxalates and urates are abundant, and the spectrum shows the appearances characteristic of methæmoglobin. Glycosuria has been observed, but is purely accidental. Atrophy of the skin and nails is often a sequel of the disease. The skin sometimes shows patches of erythema or urticaria, but scleroderma is the most striking cutaneous change in the disease. Ankylosis of joints has been described. The skin and joint troubles have not yet been satisfactorily explained. The eyes have given rise to some interesting observations. Amblyopia is not infrequent, and may be the result of some of the retinal changes to be mentioned. Myosis and retraction of the eyeball have on the one hand been described, while, on the other, mydriasis, with some degree of exophthalmos, has been observed. These appearances are attributed to disturbances of the cilio-spinal apparatus. A local contraction of the arteries and dilatation of the veins has been seen in the fundus. Cerebral attacks occur. Transient hemiplegia with aphasia has been noticed, while melancholy and mania have been present in some cases.

The symmetrical yet localised distribution of this affection and its paroxysmal occurrence render its recognition a matter of no great difficulty. No other condition presents a similar combination of character, and from such affections as chilblain or frostbite, as well as profound cyanosis, these features easily distinguish the disease. Its differentiation from erythromelalgia will be discussed in the following article.

Prognosis and treatment.—As a general rule the outlook is not unfavourable, but the prospect for each patient can only be estimated by the limitations of the environment. Even when gangrene has occurred, recovery often ensues, necessarily with some deformity. An instance of this kind in a patient, who is now above threescore years and ten, has been long under my observation.

In the milder forms, beyond attending to the diet, which should be nutritious, and giving directions about avoiding cold, no special line of treatment is necessary. In the more severe types careful management is required. Nourishing food and warm clothing are needful. Alcohol, in my own experience, is rather hurtful than helpful. The nitrites internally and massage externally are undoubtedly advantageous. Galvanism, administered as recommended by Barlow, in bath form, is of real benefit. By immersing the affected limb in a large basin of tepid water containing salt, in which one pole of the battery is placed, while the other is applied over the limb higher up, a continuous current can be employed. Barlow recommends that the strength of the current should be as great as the patient can comfortably bear, that the circuit should be frequently closed and opened, and that the patient should make voluntary movements with the muscles while the current is in action. A strength of 10 or 15 milliampères has, in my experience, been sufficient.

G. A. GIBSON.

ERYTHROMELALGIA.

Syn., Red Neuralgia.

This is a chronic disease, in which there is local flushing, heat, and pain of one or more of the extremities, increased in severity when the parts are dependent, and not showing any tendency to symmetry. Although cases of the kind were undoubtedly described by Graves, general attention was not attracted to the condition until its description by Weir Mitchell.

Etiology and pathology.—It is at least twice as common in men as in women, and occurs more frequently in middle life than at any other period. It is thought that occupation takes some part in the production of the disease—such employments as involve long standing in varying temperatures being usually regarded as important. It has been seen as a sequel of pregnancy. Previous diseases, such as malaria, appear to have some influence, while the anæmic, hysterical, and neurasthenic furnish a certain proportion of cases. Injury and long-continued local strain have occasionally been found.

Opinion is divided as regards the pathology of this affection. It is regarded as being on the one hand a "nerve-end neuritis," and on the other hand as a "vasomotor neurosis." The nerves supplying the affected regions have been found absolutely normal by Dehio. Inasmuch as intermediate cases between erythromelalgia and Raynaud's disease have been described, it seems probable that both maladies present degrees of analogous conditions, and Barlow's view appears reasonable that the affection now under consideration and the three clinical types of Raynaud's disease differ from one another in the extent of the vascular storm, and in the sequence of the spasmodic and paralytic appearances. There can be no doubt that the symptoms of angio-neurotic œdema, often regarded as deserving the title of a distinct disease, fall into the same series.

Symptoms and diagnosis.—The earliest symptom is pain in one of the extremities, which is increased when the part is allowed to hang down, or when it is employed in movement. It is relieved by rest. If the dependent position is maintained, local redness shows itself, and is characterised by the brightness of the tint. Violent throbbing ensues, and the pain increases in severity, being attended by great tenderness to touch, and increased sensitiveness to heat and cold. There is a local increase of temperature, and some swelling, without any obvious pitting on pressure. The throbbing gradually subsides, the affected part becomes purple instead of crimson, and the colour slowly fades away. Occasionally some vesicles appear during the acute stage upon the surface implicated, while hardness of the tissues, clubbing of the fingers and toes, and arching with thickening of the nails may result. Wasting of the muscles has been described, but it is slight in degree. In one case, which has frequently been under my observation, and which presents a combination of Raynaud's disease and erythromelalgia, there has often been painful contraction of the intrinsic muscles of the affected hands and feet.

It is usual to regard the following as essential points of difference between erythromelalgia and Raynaud's disease:—The former does not begin with ischæmia; it has distinct throbbing of the arteries; it has no loss of sensibility; it is attended by hyperalgesia; the temperature is increased; and there is no gangrene. In the latter there is at first ischæmia; there is no throbbing; there is anæsthesia with analgesia; the temperature is reduced; and gangrene is common.

Prognosis and treatment.—The affection may persist for many years. In one case described by Morgan, it occurred in paroxysms during a period of twenty-seven years. It has apparently but little tendency to shorten life, but it interferes greatly with its comfort and usefulness.

Abundance of nutritious food is to be recommended. Locally, the application of cold is beneficial during the attack, which may sometimes be warded off by this means and absolute rest in the horizontal posture. Barlow is doubtful as to the value of massage. The subcutaneous employment of morphine with atropine has been found of some utility.

G. A. GIBSON.

HYSTERIA.

The nervous system, apart from definite disease, is subject to various general or local states of instability or disorder, to which the terms hysterical, and of later years functional, have been applied.

The term hysteria in the present article is applied to the functional disorders of the psychic portions of the nervous system, and the term functional to similar disorders of the remainder of the system. This distinction is useful for the purposes of classification, though naturally no hard and fast line can be drawn between the groups, and this is especially the case, as not only may functional disorders be associated with hysterical manifestations, but it is by no means unusual to find organic disease of the nervous system associated with the latter.

As this article deals primarily with hysteria, the mental symptoms are consequently discussed before the physical, and in the latter the purely functional varieties occupy a subsidiary position in the description.

Etiology.—The most important factor in the development of hysterical and functional disorders of the nervous system is undoubtedly a hereditary neuronic instability. This chiefly concerns the most highly developed or psychic portions of the cerebrum, and is evident clinically as emotional rather than intellectual instability, though the latter, if less visible, probably coexists to an equal or a greater degree. This factor may be due to a general lowering of the vitality of the whole body, or may be the result of an ancestral weakness of the nervous system alone, or these conditions may coexist. In the former case the predisposing cause is such a family history as of tuberculosis, syphilis, alcohol, gout, or diabetes; and, in the latter, one of insanity, epilepsy, consanguinity, crime, or asthma. The most important evidence of the frequency, it might almost be said the necessary existence, of this hereditary influence is to be obtained from a study of the mental characteristics of the collateral relatives and parents, not necessarily the subjects of hysteria but neuropaths. Experience also shows that, of all the varieties of nervous disorder or disease, this direct evidence of hereditary predisposition is most frequently to be obtained in cases of emotional instability of the nervous system. This may probably be explained by the hypothesis that, whilst this condition is the least serious of nervous affections, it is also more readily produced and more easily prevented than the more grave mal-developments and diseases of the nervous system.

The next factor in the production of the disorder under consideration is the environment of the patient. Probably a large number of the cases of hysteria would not develop, at any rate to a marked degree, were it not for injudicious treatment on the part of their relatives. The parents of hysterics are in many cases the most unsuitable guardians for the patients that could well be discovered, and are unwittingly though largely responsible for the unhappy results which frequently follow the development of the disease. Many other factors concur in the production of the particular symptoms from which the subjects of hysteria suffer. In many cases imitation is a potent cause—as when a girl nursing her paralysed father develops paraplegia, or a child lying in the next bed to a case of Pott's disease becomes unable to stand or walk. Other and more common instances are the development of hysterical attacks, religious ecstasies, wryneck, winking, stammering, retention of urine, etc., which frequently occur as a result of imitation even in apparently normal persons. It is well known, too, how readily mannerisms and tricks of speech are imitated by children, in whom the nervous system, owing to its immature condition, is peculiarly susceptible to external impressions.

In other cases the result is due to a former injury, overstrain, or disease of particular regions of the body, especially the limbs, which draws attention to the part, and, acting by suggestion on the unstable nervous system, produces various local disorders, probably of psychic origin. Again, non-psychic functional disorders may be the result of the overwork of certain groups of neurones, as in the cramps developed by clerks, typists, or musicians, in which cases the cause is most probably a Rolandic instability, from excessive performance of the particular voluntary movements concerned.

To the above may be added various kinds of shocks to the nervous system; the general lowering of potential due to over-work, privation, or disease; and the psychic effects produced on the unstable nervous system by irregular habits and abuse of the various bodily functions.

Of the more personal factors in the production of hysteria, the chief are sex and age. This is probably partly due to the naturally less stable nervous system of the female sex, and partly to the difference in the education of the two sexes. In the lower classes, where little or no difference exists in this latter respect, hysteria, or its congener hypochondriasis, is probably equally common in the two sexes, but in the upper and middle classes it is certainly more common in the female. This is probably due partly to the absence of healthy occupation, partly to the barriers set up by social custom, which so markedly limit the activities of women, and partly to ignorance of sexual subjects, and in older women to repression or perversion of the sexual instinct, and particularly to any cause, surgical or otherwise, which may direct their attention to the reproductive organs. As regards age, the type of the disorder varies at the different stages of life, and its production is accelerated by the various conditions of stress to which the nervous system is subject—for example, at puberty and the climacteric period. Hysteria occurs at times in quite young children, leading in them to impulsive acts and to the development of functional disorders. In girls, emotional disturbance and the more obvious hysterical disorders are the chief characteristics; whilst in young men, though this form occasionally occurs, the tendency is as a rule in the direction of depression, especially with reference to the general health or the sexual functions. Finally, in older people of both sexes, hypochondriasis in its various forms is the chief characteristic, and these cases are, as might be expected, particularly intractable as regards treatment, and especially troublesome in club practices and in the out-patient departments of general hospitals.

Symptoms.—**Mental.**—The mental disturbances in hysteria are occasionally slight and relatively unimportant, but they are nevertheless on careful study as characteristic of the disease as the more obvious physical symptoms.

The chief mental symptom of the disease, as might be expected from the definition given earlier in this article, is emotional instability. This agrees with the relative frequency of the disease in women, in whom the nervous system is, as a rule, less stable than in men, and also the relative frequency of functional disorders of the nervous system in the former sex. The patient readily becomes excited or depressed, and the emotions are exaggerated and rapidly get beyond control. They are frequently abnormal in nature, the patient laughing when she might reasonably be expected to cry, and *vice versâ*. This condition is associated with increased sensitiveness and an excessive reaction to external impressions. The power of concentrating the attention except on personal matters is, as a rule, deficient, and may be markedly so. The memory is frequently normal, but almost as frequently impaired: and sometimes the events of short or in some cases even long periods of time are absolutely forgotten. The intelligence is as a rule good, and in many cases is very acute: but the judgment is weak and extremely liable to be influenced by the prevailing emotions. The will power is usually deficient, and the patients are generally inert and lazy during the inter-paroxysmal periods, but they are not infrequently energetic, mischievous, and troublesome to their friends. Self-consciousness, especially in the more severe cases, is exaggerated, the patient concentrating her attention on her ailments to the exclusion of her duties or the care of her health. There is, in consequence, frequently a desire for sympathy and notoriety, which leads the patient to be unwittingly deceitful and even untruthful and dishonest. She is often selfish and suspicious, even quarrelsome and irritating to those with whom she comes into contact. She is, however, quite conscious of the impropriety of her behaviour, and during the intervals of reasonableness she may apologise, even abjectly, for her misconduct.

This condition grades insensibly into the milder varieties of "moral" insanity met with in asylum experience. These patients are usually girls of 15 to 20 years of age, and are generally found in asylums, owing to the fact that their friends find them utterly unmanageable at home. From a study of the mental characteristics of their parents, and from the fact that kind but firm treatment keeps these patients in a perfectly stable condition even for months at a time, during which they work well and are medically sane, the conclusion that the home atmosphere is largely responsible for their unhappy condition cannot be avoided. Whilst these patients are truthful and tractable, under those who understand them and are kind to them, they are liable to outbursts of uncontrollable excitement from slight causes. They develop violent affections and dislikes, and occasionally, with little or no provocation, and frequently from pure mischievousness, and probably also largely from imitation, they tear their clothes, smash the windows, and attack nurses and patients whom they dislike.

Similar but more severe examples of the same class of case occur in rather older girls, who, owing to unhappy home surroundings, have probably taken to drink, gone on the streets for a time, and then been brought to an asylum as the result of a street brawl. The attack of insanity may be anything—from extreme uncontrollable excitement to acute mania, of longer or shorter duration, after which the patient is in the condition above described, the prognosis being, of course, graver than in the previous cases, owing to the dissolute life and consequently diminished moral sense of the patient.

Such patients may secrete glass, needles, and other articles, and produce them at a favourable opportunity, in order to get their attendants into trouble; they may smash, tear their clothes, and attack those around them; they may be deliberately indecent, or degraded in their habits; they may injure themselves with anything they can obtain for the purpose, for example, by thrusting pins or needles under their skin, or into their noses and ears, or by swallowing them; they may attempt suicide, sometimes seriously, sometimes in pretence; and they are generally a terror to all those with whom they come into contact. On the other hand, they frequently have a strong sense of honour, and if they make a promise to behave themselves to some one they respect, they may be trusted to an almost unlimited extent. It is almost unnecessary to add that these conditions are not attended by any marked degree of mental impairment, and that the case only ends in death from accident or intercurrent disease. In all these varieties of mental instability, the attack of excitement may be preceded by a period of malaise, by an attack of dyspepsia, or by a referred pain of visceral origin, and at these critical periods the slightest cause may precipitate its onset. In this point they show their relationship to the more common emotional disturbances of hysteria, as also in the not unfrequent occurrence of definite hysterical symptoms, as for example astasia and abasia, etc. Similar examples of the same class of case, but usually in older women, are often to be found in the chronic wards of asylums. The characteristics of these cases is emotional instability, with liability to severe attacks of excitement, and without any obvious mental enfeeblement.

In the same group as above may probably be placed a certain number of the cases of sensitive children, who, owing to a harsh reprimand, or to a refusal of what they consider a reasonable request, lose their self-control, and attempt to hang or drown themselves. In older persons, also, many cases of unpremeditated suicide may probably be attributed to a similar condition of mental instability associated with a shock beyond their endurance; as when a hard-working woman returns home at night to find her furniture sold, and her husband squandering the proceeds in drink, and, after an altercation with him, at once throws herself into a neighbouring pond, which may or may not contain enough water to drown her.

In a different group should be placed a number of cases, usually of women of middle age, and not necessarily unmarried, who may become mentally unstable and strange in behaviour, or may develop a more or less severe attack of insanity after abdominal operations, especially ovariotomy. In these cases ovarian and mammary hysterogenic zones may be present. The patients are usually extremely proper in their behaviour, they may strongly object even to ordinary medical examination, and they may even for years conceal from their medical attendant that they have had an operation performed. They are usually quiet and well-behaved, but are mentally unstable, emotional, and old-maidish even when married. The periodic attacks may be preceded by pain, or sensations in the head, or by pain in the region of the heart, or in the abdomen. If attention be paid to their complaints, these become more severe, and their medical attendant is always in requisition. If their symptoms, on the other hand, are made light of, the reverse may happen, though frequently they lose confidence in their adviser; and if not in an asylum, they try in order all the practitioners in the district, and are treated in turn for hysteria, cardiac disease, dyspepsia, etc., according to the attention paid to their complaints, or to the degree of examination they permit.

Lastly, a similar group may be made of older or senile cases, also usually females, whose marked characteristics are extreme nervousness, and a concentrated attention to their ailments. These patients are usually depressed, lachrymose, and miserable, and are never tired of detailing their symptoms, and of varying the disease from which they suffer. They complain of feelings of weight and tension, and of hot and cold water flowing through their head, of various pains and sensations in the cardiac region and in the abdomen, and especially of a fluttering of the heart, probably diaphragmatic in origin. They go into trances at night, and feel their feet and legs and arms drawn and cramped, without ability on their part to resist, and frequently complain of a feeling that they are about to die, which is probably an exaggeration of the normal sensation felt by ordinary persons when they are about to fall asleep, but are for some purpose endeavouring to keep awake. These patients state that they dread bedtime, owing to the agony they suffer when sleep comes on, as they are convinced that they will never awaken again. They piteously ask their attendant whether he really thinks they can get better, and in turn ask whether they are suffering from softening of the brain, heart disease, or cancer. They state that they are too miserable and weak to speak or stand, yet they brighten up during conversation, and under the influence of suggestion will walk perfectly well and quite strongly, although all the time they continue to repeat that they know they cannot stir a step. The effect, however, soon passes off, and the next day they are again in the same condition. It would be impossible, however, to detail all the symptoms from which these patients suffer.

This group of cases is exactly analogous to the cases of hypochondriasis in men, which will be alluded to in a separate paragraph, and were it not customary to describe such cases under a different name, no further description would be needed. These cases, if well-to-do, are usually kept at home, but if without means they drift into workhouses and asylums. In an exaggerated form the same type of case is met with commonly in asylum experience. Here, however, the patient often suffers from a definite delusion. She has no head or body, her throat or lower bowel is blocked up, or she has a worm in her stomach. These cases are otherwise exactly like those above described. It is, however, by no means rare in such cases, where a fixed idea of disease is present, to find, post-mortem, that the idea had an organic foundation in visceral disease. In other cases a sham operation to remove the worm or other cause of trouble, provided it be slightly painful, will by suggestion, at any rate for a time, cause the delusion to disappear, though if an organic cause for the discomfort exists it is certain sooner or later to reappear. These cases consequently require most careful examination, in order if possible to eliminate the presence of visceral disease.

From a review of these groups, it seems evident that, the first is chiefly characterised by the prominence of mental instability, and the last by that of mental perversion, whilst the second occupies an intermediate position between the two. In all, however, the two characteristics of hysteria, namely, mental instability and perversion of the normal cerebral representation of sensation, are present. It is consequently probable that the usually described rarity of hysterical manifestations in the insane is only correct in so far as it refers to the confusional, chronic delusional, and demented cases which make up the majority of the inmates of asylums, and in whom the existence of purely functional instability of the nervous system, without any neuronic degeneration, is of extremely rare occurrence.

In the ordinary form of hysteria, certain mental symptoms, closely related to those occurring in insanity, occasionally occur. Amongst these is the incidence, described by Charcot, of periods of sleep lasting for days or even for weeks, during which slight consciousness exists. If food be presented to the patient when in this condition, she eats and drinks, and at times even shows preferences for particular articles of diet. Another symptom of rare occurrence is a sudden loss of consciousness, during which the patient acts as usual, transacts business, and even performs journeys; and on awakening is at a loss to explain how he happens to be where he finds himself, and is quite unconscious of what has happened in the interval. Examples of this condition are recorded by Charcot and by Wilks, as for instance the case of the miracle at Lourdes, where the patient later on denied his identity, and stated that the sufferer was his brother. This condition is probably related to the lapses of memory previously referred to, and to the double consciousness which exists in certain cases of epileptic and ordinary insanity. At times, in ordinary cases of hysteria, maniacal attacks, coexisting with definite delusions, may occur and last for a considerable time. Of less moment are the transient delirium and hallucinations which occasionally follow hysterical seizures, and the mental auræ which not unfrequently precede them.

Patients suffering from hysteria are occasionally accused of wilful simulation, dishonesty, and imposture, but on quite insufficient grounds They are really quite unable to help their failings, and during their lucid intervals they are frequently most anxious to find a means of cure. The few cases of obvious imposture which have been noted are of a criminal rather than of an hysterical nature. Many cases of apparent imposture, on the other hand, where the patient denies that he has experienced what is known to have happened to him and even his identity, are examples of temporary insanity.

In our present state of ignorance of the causes of the mental manifestations of hysteria and of insanity, it is probably premature to generalise on the nature of the relationship between the two conditions. It is, however, perhaps permissible to suggest the possibility that hysteria, and the various hysterical states above referred to, may be considered as functional disturbances of the psychic processes, whilst in true insanity the psychic disturbances may possibly be always associated with definite neuronic degeneration to a greater or a lesser degree. This is evidenced by the fact that the majority of the true insanities result invariably in a greater or lesser degree of dementia, whilst in the majority of the hysterical cases above referred to any obvious degree of dementia is rare. This hypothesis being postulated, it is only necessary to add that whilst functional disorder of any part of the nervous system does not preclude the coexistence of organic disease, so hysterical disorder of the psychic regions of the cortex may be equally associated with definite neuronic degeneration and true insanity.

It is now necessary to turn from the mental symptoms of hysteria which appertain to functional disorder of mental states and processes, to the more obvious and more common symptoms of the disease which appertain to functional disorders of the regions concerned in the cerebral representation of bodily sensations and functions. These symptoms, as will be seen on reference to the first paragraph of this article, must be grouped under two categories, the hysterical and the functional. In the purely hysterical group, the sensory or motor affection exists in a part of the body as the patient knows it, whilst in the case of the functional group the disability depends for its distribution and degree on the particular group of neurones affected. From considerations of space, and owing to the fact that the symptoms are detailed at length in the following account, it is unnecessary to give examples of these two classes, as they will readily be noticed by the reader. It may, however, be worth while to refer, as a very obvious example, to the disorder of vision met with in hysteria. Here, of course, the patient is quite unaware of the hemiopic projection of the retinæ in the calcarine fissures, and consequently the disorders of vision are never hemiopic but invariably amblyopic. In other words, the patient suffers from a more or less marked loss of vision in one or both eyes, viewed as eyes. It is probable, however, that the symptomatology of hysteria is frequently complicated, particularly in educated patients, owing to the fact that they often carefully study their cases and read up the diseases of the nervous system, until the resulting complex of symptoms which they describe is, to say the least, extremely bewildering. In such cases the existence of emotional instability, together with the fact that the symptoms described are so heterogeneous as to be inexplicable as results of any possible organic lesions of the nervous system, usually enables a diagnosis to be made. Occasionally, however, a case is met with which presents so much difficulty, that it is absolutely necessary to treat it as one of organic disease.

The objective symptoms of hysteria will be grouped as—(*a*) ordinary symptoms, and (*b*) hysterical attacks.

Ordinary symptoms.—The pains of hysteria, apart from hyperæsthesia, require little description. They may occur anywhere, quite apart from nerve distribution, may begin suddenly and cease as suddenly, and are as a whole quite excessive in degree. They are apt to cease when the attention is distracted, and are increased by sympathy and inquiries. They also occur without any local signs of disease, and if localised are unaccompanied by the normal segmental areas of hyperæsthesia. These pains are probably of considerable importance, if we understood their significance, with reference to the cortical representation of sensation, concerning which our knowledge at present is in its earliest stage.

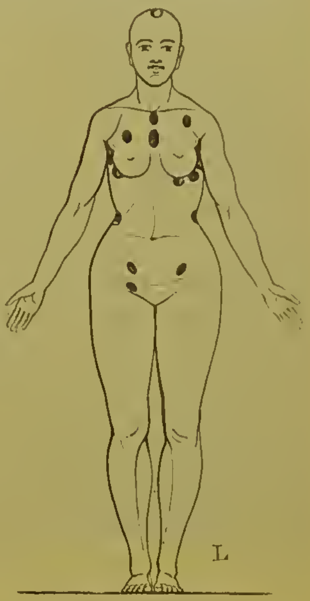

Fig. 25.—Hysterogenic zones on the anterior surface of the body. —After Charcot.

The hyperæsthesia of hysteria is less frequent, less permanent, and less extensive than the anæsthesia to be later on referred to. It may be extreme or may be unknown to the patient. It may be cutaneous, subcutaneous, or extend deeply, and is usually more marked on the left than on the right side. At certain points the hyperæsthesia is excessive, and light friction over these regions will start other hysterical manifestations, or even definite hysterical attacks, whilst pressure on them during an attack may stop or modify it. The chief hysterogenic zones on the body are illustrated in Figs. 25 and 26. They are very variable in distribution and extent in different persons. They exist also on the limbs, according to Pitres and Gaube, but here the zones are exclusively excitant, and pressure on them during an attack has no effect on its course or severity. The most important of these, the ovarian, is situated in the left iliac region. Its presence is not necessarily restricted to women only, though in men the corresponding zone may be in the inguinal region. Others are situated under the left breast, over the crests of the ilia, in the epigastric region, over the sternum, below the clavicle, in the neck, over the left temple, and on the vertex of the head; also posteriorly, below the scapulæ, and over the upper and lower dorsal spines. Tenderness over the vertebræ, especially over the cervical or upper dorsal spines, is not uncommon, especially after accidents. It is acute, excessive, and superficial. Tenderness of the breast, usually the left, is less common. It may follow a blow, and painful lumps may exist, but as a rule physical signs are absent. It is an exaggeration of the tenderness of the hysterogenic zone in this region, and may cause considerable disturbance in the patient by suggesting cancer of the mamma. Tenderness in the region of the joints, not unfrequently following an injury, the hysterical coxalgia of Brodie, is not uncommon. It is most common in the hip, knee, or ankle, and the pain on

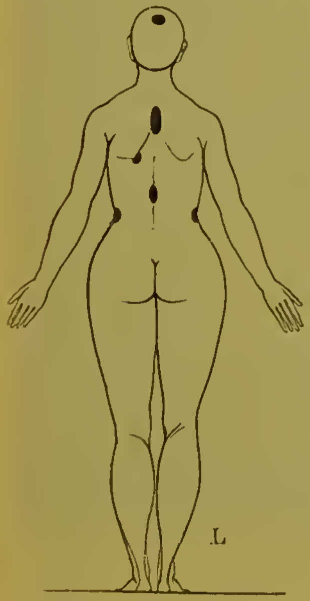

Fig. 26.—Hysterogenic zones on the posterior surface of the body. —After Charcot.

pressing up the knee or heel is superficial and not limited to the joint, but extends above Poupart's ligament and to the lower part of the abdomen and buttock. The patient is not awakened at night by muscular jerks. There are no physical signs under chloroform, but there may be muscular wasting or vasomotor changes.

The anæsthesia of hysteria is a very frequent symptom of the disease. It usually exists as a single (or sometimes double) hemianæsthesia, including the mucous membrane of the back of the pharynx, the conjunctiva, and the special senses; and the muscular sense may be affected. The latter, in general, is completely lost in the anæsthetic area, but may be more or less preserved. If less extensive in distribution, the anæsthesia is not necessarily limited to the periphery of a limb, but may exist as sleeve-like patches, occupying, for example, the forearms or thighs only, or it may appear simply as irregular islets over the body. It usually extends in the case of paralysed joints for a short distance above the affected joint, and is limited sharply by a circle round the limb. It may come on suddenly or gradually, may vary from time to time, and may even be unknown to the patient, especially if touch is relatively little affected. With reference to the psychic origin of these disorders, attention may be drawn to the fact that, when unilateral, it much resembles, excepting in its greater intensity, the kinæsthesia present in lesions of the Rolandic area. In these cases, in addition to a more or less marked loss of the kinæsthetic sense, the power of localising touches is usually deficient, the patient, for instance, referring a touch on the wrist to the middle of the arm, and one on the middle of the arm to the elbow. This condition was pointed out by Horsley as a characteristic of lesions of the Rolandic area, and it is well marked in many cases of functional disorders of this region.

In the account given by Head of hysteria and of functional disease, in hysteria, touch, pain, heat, and cold are absolutely coterminous; whereas in non-psychic functional disease, sensations of pain and temperature are more affected than those of touch. In the former, also, the reflexes are unaltered over the anæsthetic and analgesic areas, whilst in the latter they are abolished.

The anæsthetic areas may frequently be transferred to the opposite side of the body directly by suggestion, or indirectly by the application of such agents as magnets or metals.

The anæsthesias of cortical origin, whether due to functional disorder or organic disease, are necessarily combined with paralysis of the corresponding portions of the limbs concerned. This was originally pointed out by Bastian, who first propounded the view that the so-called motor areas of the cortex are not really motor in function, but are the regions of the cortex in which tactile and muscular sense impressions are stored. The term kinæsthesis was introduced by Bastian to describe this function, and he pointed out, or rather insisted, that the paralysis resulting from loss of function of this part of the cortex is due to inability to ideate the movements which it is desired to perform—which movements are acquired chiefly or entirely as a result of education or voluntary imitation.

Disorders of the special senses.—*Sight.*—Complete blindness is rare in hysteria, but some degree of amblyopia is very common. The amblyopia is usually one-sided, and, according to Pitres, exists in monocular vision only, disappearing during binocular vision. Hemianopsia and central scotomata never exist in pure hysteria. There is no decrease in the acuity of vision, but photophobia is common. Colour vision may be entirely lost, everything seen appearing grey, a condition of pure achromatopsia. Concentric contraction of the fields of vision is a fairly common condition, and whilst it is usually most marked in one eye, it is generally, as was first pointed out by Landolt, present to some extent in both. If this contraction exists to a marked degree for white light, it is not unusual to find the red field more extensive than the white. The amount of contraction varies in individuals, and may be to a few degrees only, or central vision alone may be left (see diagrams in Fig. 27). When there is affection of colour vision, the contraction, according to Charcot, from the least to the best preserved colour, is in order—violet, green, blue, yellow, and red, or more rarely violet, green, red, yellow, and blue. During perimetric examination, fatigue readily results, and the observations soon disagree unless they are rapidly conducted. Thus the earlier observations give a larger field than the later, unless an interval for recuperation is allowed. Charcot describes a condition he calls monocular polyopia. In this state the patient, with one eye closed, sees two objects instead of one, and one of these is usually larger than the other. There may also be micropsy or megalopsy, the object seeming smaller or larger than its actual size, but there is often one point at which the object seems of natural size. In these cases the conjunctiva is always more or less insensitive. The condition is explained by Charcot as due to spasm or paralysis of the ciliary muscle, together with irregularities in the refraction of the lens.

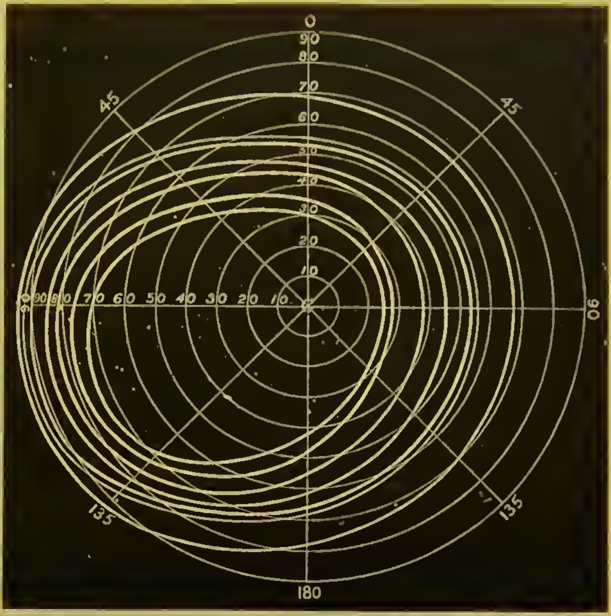

Fig. 27.—Visual field of the normal left eye. The areas marked represent from without inwards the fields for white, blue, yellow, orange, red, green, and violet light.—After Charcot.

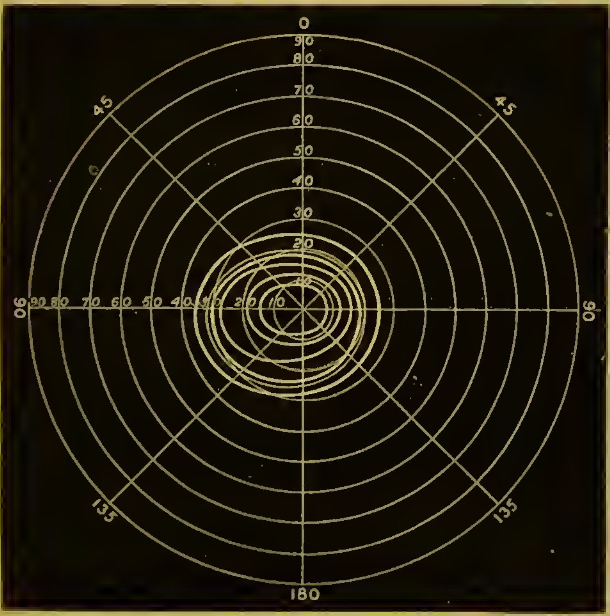

Fig. 28.—Visual field of the left eye in a case of hystero-epilepsy with left hemianæsthesia. There is concentric contraction of the field. The areas marked represent from without inwards the fields for white, blue, yellow, red, green, and violet light. Acuity of vision $\frac{1}{20}$.—After Charcot.

Finally, certain symptoms may be mentioned here, namely, the subjective visual sensations or auræ which may precede hysterical seizures, and the visual hallucinations which occasionally occur during the final stages of these attacks.

Hearing.—Relatively little has been published on the condition of the sense of hearing in hysteria, but hysterical deafness is stated to exist, and to be usually combined with mutism. The patients, however, though deaf and dumb, can read and write perfectly. In these conditions, as in the case of sight, more or less anæsthesia of the external auditory meatus, the membrana tympani, and the pinna occurs. As in the case of sight, noises in the head or ear and vertigo may exist as auræ, and auditory hallucinations are not uncommon in the later stages of the hysterical seizures

Taste, smell, touch, and muscular sense.—Affections of these senses come under hysterical anæsthesia, as a part of this condition, and do not require a separate description.

Motor.—*Spasms and contractures.*—Spasms and contractures frequently exist in cases of hysteria, and some of these are much more common than others. For the particular variety exhibited in each case there is probably an inherited or acquired cause, which we are unable to discover owing to the present limitations to our knowledge, for the localised neuronic instability. It is impossible here to say more than that these particular symptoms of hysteria are peculiarly liable to develop as a result of imitation.

Tonic spasms.—The most common forms of hysterical tonic muscular overaction are spasms and contractures of the arms and legs. These may last for months or even for years, and in many cases until the condition, without an operation, is permanent, owing to fibrous adhesions in the joints, and definite shortenings of certain of the contracted groups of muscles. The arm is usually bent at the elbow, and the wrist is flexed and adducted, the fingers being firmly folded over the flexed thumb, occasionally to such a degree that the nails are driven into the palm. In the case of the lower limb, the leg is extended with the toes pointed, though several varieties of club-foot may occur. The reflexes may be unaltered, absent, or increased, and a spurious ankle-clonus may be obtained. These conditions are permanent even during sleep, and are not resolved during anæsthesia until the highest degree is reached. Charcot has pointed out that these contractures differ from those of organic origin in the relative suddenness of their onset, and owing to their liability to pass away rapidly, or even to come and go several times.

Other less frequent varieties of tonic spasm may occur in hysteria. One of the most common of these is the phantom tumour, due to the irregular tonic spasm of the abdominal walls, which occurs in some cases of flatulent dyspepsia. A glosso-labial hemispasm is also by no means rare. It is necessary to draw special attention to this variety, owing to the possibility, on casual examination, of mistaking it for facial paralysis of the opposite side. If the tongue be carefully examined, the deviation is obviously due to spasm, as the tongue on the side to which it points is relatively thick and rounded. There is also no paralysis of the side not affected by the spasm, and during a forcible blowing movement there is no bulging out of the cheek, but rather a retraction of the buccal muscles.

Tremors and twitchings.—Clonic spasms of the neck and shoulders are far from rare in hysteria, though a tonic spasm of the neck is perhaps more common. Twitching of the eyelids also occurs relatively frequently, and this may be more or less permanent, or may come on at intervals. Three varieties of muscular tremor have been described. Any tremor may be simulated, but the most common one is very rapid and fine, and occurs usually in the hands, but also in the tongue and head. Very common, also, is the occurrence of irregular tremors, like those in disseminated sclerosis, which are absent when the patient is at rest, but which come on during movement. They seem to be an exaggeration of the usual tremor of nervousness. The third variety is less common than the others, but is much more remarkable. It consists of large, slow rhythmic movements, which may involve the whole body or be limited to the wrists or elbows or to the lower extremities. The chief characteristics are extreme regularity in the range and rapidity of the movements, combined with constant repetition. Varieties of these movements are the salutatory, saltatory, and hammering spasms, which are described by Charcot as occurring during the hystero-epileptic attack. They may be decreased or stopped by distracting the attention of the patient, or by causing her to occupy her muscles in other movements. They may also be suppressed or modified by pressure on one of the hysterogenic zones. According to Pitres, they cease during sleep. These movements, frequently described as hysterical chorea, are probably related to those occurring in the dancing epidemics of the Middle Ages, in which the movements were also of a slow and rhythmic nature.

Of the functional spasms of cerebro-spinal rather than psychic origin, various degrees of spastic monoplegia and paraplegia are the most common, and, except for the facts that they may be intermittent, and that there are no physical signs pointing to an organic cause, *e.g.* vertebral disease, they are difficult to distinguish from spastic conditions due to organic disease causing descending degeneration of the pyramidal tracts. Charcot drew attention to the extreme degree of the rigidity in some of the cases, and stated that, in his opinion, after a certain period, an actual sclerosis of the lateral columns would result.

Paralysis.—Amongst the paralyses of hysteria a rather rare but obviously cortical variety is of great interest. This condition is described by Charcot under the terms astasia and abasia. The patient can move her legs in a co-ordinated manner whilst lying in bed, and the muscular strength is apparently normal, but she is quite unable to stand upright or to walk.

The most common of the hysterical paralyses are hemiplegias, without affection of the face, as was first pointed out by Todd, and paraplegias; but any monoplegia, though these are rare, may exist. The varieties of paralysis to be now referred to, being of cortical origin, are flaccid in character, and are associated with profound loss of muscular sense, and with some loss of tactile and general sensibility. In these cases the cutaneous anæsthesia forms a zone corresponding to the paralysed limb, and is limited above by a circle somewhat higher than the joint affected by the paralysis. It differs from the cerebro-spinal anæsthesia of organic disease, owing to the fact that in the latter the anæsthesia never ends above as a circular line, but is related to the cutaneous distribution of the affected sensory nerves. In the hysterical paralyses there is no reaction of degeneration, no loss of reflexes, and if any wasting occurs it is generally distributed over the paralysed parts. The limb may also be blue and puffed, this condition being described by Charcot as the blue œdema of hysteria. Hysterical paralyses are also characterised by an entire absence of effort to move the limbs on the part of the patient. The arm or leg is carried or dragged, as if it did not belong to the patient but was fastened on to her; and in the case of the leg the circumduction performed by hemiplegics to get the foot above the ground is not resorted to. If the limb also be suddenly and forcibly moved, a certain amount of resistance may be perceived. If facial deviation occurs, it is invariably due to glosso-labial hemispasm. It may be added that in hysterical hemiplegias, whilst retention of urine is common, it is rare to meet with incontinence of urine or fæces.

Functional aphonia is very common in hysteria, but other varieties of local paralysis, such as ptosis, are of relatively rare occurrence. In aphonia the patient can whisper quite well without hoarseness, but cannot close her glottis for voluntary phonation, though she can usually cough quite loudly. Very frequently the aphonia is associated with a mild attack of laryngitis. The larynx is easy to examine, owing to the coexisting anæsthesia of the fauces. Of all the symptoms of hysteria, this is probably the one which is perhaps the most amenable to treatment. Hysterical mutism has already been referred to under affections of hearing.

Visceral disturbances.—*Circulatory.*—Of the circulatory disturbances occurring in hysteria, palpitation, rapid or tumultuous action of the heart, is by far the most common. This may be combined with throbbing of the abdominal aorta and of the superficial vessels, especially in the neck. There is, however, none of the paroxysmal pain of aneurysm. There may also be an extreme, and more or less permanent, rapidity of the pulse. The superficial vessels may be so sensitive as to yield in local irritation of the skin an artificial urticaria, frequently described as autographic skin. On the other hand, it is not unusual to meet with syncopal attacks combined with decreased frequency of the beat of the heart. This especially occurs in catalepsy and trance. There may also be a stifling feeling or sensation of suffocation with an almost imperceptible pulse.

In the analgesic regions of hysteria vasomotor spasm is common, and on pricking these parts little or no blood can be obtained. Associated with this condition there is frequently extreme sensitiveness to cold. The patient again may be alternately red and pale, from alternate dilatation and constriction of the cutaneous vessels, and may suffer, especially in the limbs, from alternate feelings of heat and cold.

There may be curious sensations about the heart, and even attacks resembling angina pectoris. This symptom may occur at any age, it often comes on periodically at night, it is more frequently caused by emotion than by fatigue, and it may be associated with the presence of hysterogenic zones in the præcordium or the mid-dorsal region of the spine. The attacks last longer than ordinary anginal attacks, and, according to Charcot, are generally associated with symmetrical asphyxia of the extremities. The chief symptoms are a sense of distension, to the extent of suffocation, combined with severe emotional disturbance, and the pain is less severe in character and is usually situated at the back of the head and neck. Hæmorrhages of various kinds are of by no means rare occurrence. They may occur superficially, from the skin or mucous membranes, as in spurious hæmoptysis, which is probably generally caused by the sucking of cuts and bites, or the patient may suffer from sore gums or epistaxis. The hæmorrhage, on the other hand, may be from internal organs, occasioning hæmatemesis and melæna, or metrorrhagia. In all these cases extreme care is necessary to eliminate imposture, as patients have been known to show to their medical attendant the blood of mammals or birds, stating that they have vomited or passed it.

Respiratory.—A hysterical cough is one of the most common of the respiratory symptoms. The cough is excessive, violent, loud, rhythmic, and monotonous, and only ceases during sleep. It is most common in young persons. Fits of laughter and weeping come under a similar category, as do sneezing, hiccough, yawning, and the making of curious noises like the cries of animals. Adductor or bronchial spasms, in attacks like those of asthma, but associated with considerable emotional disturbance, are fairly common. The patient may also suffer from dyspnœa or tachypnœa, the latter resembling the breathing of acute lobar pneumonia.

Digestive.—Hysterical anorexia, the *anorexia nervosa* of Gull, is an occasional and often grave condition. The patients will not eat, in many cases because they are absolutely without appetite; or they may eat secretly, though they absolutely refuse food at meal-times. They may, however, really not eat, and may consequently be reduced to the last stages of exhaustion from starvation, if they are not forcibly fed. Many patients suffer from periodic or almost continuous vomiting, of either a voluntary or an involuntary nature. They may be unable to keep food down for months, at once bringing up all or apparently all the food they swallow, either voluntarily or owing to spasm of the walls of the stomach of nervous origin. They may, however, eat in secret, and in any case it is probable that they do not really reject everything they swallow, but absorb enough to keep them in fair health. It is probable that in this group should be included certain of the cases of uncontrollable vomiting which occur during the early months of pregnancy.

An excessive secretion of gas in the stomach and intestines frequently occurs, and is accompanied by noisy eructations, often associated with the swallowing of air, and by loud rumblings in the abdomen. The coils of intestine are visible and very irritable, and this condition, with irregular contraction of the abdominal walls, is often responsible for the appearance of phantom tumours. Constipation is very common in hysteria, and it may be intractable, the patient only evacuating her bowels under the influence of strong aperients. On the other hand, Weir Mitchell describes a condition of irritability of the rectum associated with small and frequent stools.

Secretions.—Alterations in the amount and composition of the urine are fairly common in hysteria. On the one hand, a condition of oliguria or even anuria may exist, and, on the other, the secretion of excessive quantities of pale watery urine is by no means uncommon. The condition of anuria or oliguria has been minutely studied by Charcot. He states that the amount of urine may be markedly decreased, even for considerable periods of time. If this decrease is extreme, vomiting frequently ensues, and the vomit has been found by Charcot and Marie to contain carbonate of ammonium or urea. Under the same circumstances, the patient may suffer from diarrhœa, the stools containing carbonate of ammonium.

Retention of urine, or, on the other hand, frequency of micturition, may occur. The retention may be due to anæsthesia of the bladder or to spasm of the sphincter, or it may result from imitation. The frequency of micturition, when present, is probably due to irritability of the bladder, and it is usually associated with suprapubic pain.

Irregularity of menstruation is not uncommon, and vaginismus is not a very rare symptom. The view of Charcot, that hysterical subjects are less sexual than normal ones, is probably correct. These patients may apparently possess the sexual instinct, if men they may suffer from spermatorrhœa, but they are unable to perform the sexual act. At the same time, they may desire to marry, or they may take a great interest, from a theoretical point of view, in erotic subjects and literature. Galactorrhœa occasionally occurs, and rare cases have been described in which it existed to an extreme degree. Sialorrhœa is perhaps a more frequent symptom than galactorrhœa, and in some cases the amount of saliva secreted is very large.

Temperature.—Very high temperatures have been recorded in hysteria, but these are probably to be accepted with great reserve, for so many of the cases must be impostures, the temperature recorded being incompatible with life. With reference to the hyperpyrexias, it is probable, as Hale White suggested, that the records are due either to erroneous observation or to fraud, for recent investigations on the changes taking place in nervous tissue during high fever tend to show that a temperature of 116° would cause precipitation of a neuro-globulin in the nerve cells, and hence immediate death, whilst a temperature of only 109° or 110°, if continued for some time, would have the same effect.

Trophic changes.—Various kinds of bullous eruptions, similar to herpes, have been described by Charcot as having been produced by suggestion. These conditions are probably marked degrees of the fairly common irritability of the skin which results in artificial urticaria. Charcot has described a cold blue œdema of the extremities in many cases of contracture and paralysis, which may be associated with a spontaneous shedding of the nails. A moderately marked degree of œdema is not uncommon. This condition is most marked in the morning, and it does not pit on pressure. In many cases it is probably the result of severe neuralgic pains, especially where it is localised in one limb only. Fibrous contractions of the joints in cases of long-standing spasm have already been referred to, as has also the muscular atrophy which occasionally occurs and in which the electric reaction is normal.

Hysterical attacks.—*Simple: Hysteria minor.*—The simple hysterical attack is usually preceded by such symptoms as headache, giddiness, a sensation of suffocation, or a sudden feeling of heat. There may also occur a feeling of constriction in the throat with swallowing movements, as if the patient were trying to get rid of an obstruction (cf. the globus hystericus), or the patient may suffer from palpitation, or a feeling of constriction in the head and a beating in the temples. There may be some emotional display, as for example laughing, crying, or yawning. The commencement of the attack is then ushered in by a choking cry and perhaps a twitching of the eyelids, and the patient begins to struggle usually in a purposive rather than in a convulsive manner. She rarely falls down, but may throw herself on the ground. The tongue is not bitten, the urine and fæces are not passed, and there is, as a rule, no loss of consciousness. The facial expression again is not lost, and the eyes are generally closed. There may be such convulsive conditions as opisthotonos, and if she be restrained the patient may struggle more strongly and try to bite. As a result of the violent exertion, the patient pants for breath, and then gradually ceases to struggle, and her condition passes into one of simple emotional disturbance. She may weep and sob, or laugh in a meaningless manner. There is frequently considerable eructation of wind at this stage, and usually a large amount of pale urine is passed after the attack.

The attack is not followed by a period of stupor, but ends with the emotional display. An attack seldom occurs at night, and never when the patient is alone.

The attack may consist simply of emotional display without a definite seizure, or, on the other hand, the seizure may be more severe, and all grades exist, up to the "hysteria major" about to be described. Instead of the relatively slight prodromal symptoms already referred to, there may be a definite aura, consisting of an intense sensation in the ovarian region which passes through the præcordial region up to the throat and head; or, on the other hand, the sensation may begin in any of the hysterogenic zones, or in some cases in the limbs, particularly if some more or less permanent functional disorder exists there already. Probably the particular aura occurring in the different cases is originally due to some former overstrain, injury, or disease, or to some inherited instability of the region of the nervous system in relation to the part in which the sensation first occurs.

Hysteria major: Hystero-epilepsy.—One of the most complete and minute accounts of this condition is that of Richer, who has considerably elaborated the description given earlier by Charcot. The following is the general course of a hystero-epileptic seizure:—The attack begins, after a period of malaise, with a definite aura, after which there is an epileptic seizure followed by a hysterical attack, the latter of which consists, in succession, of a series of contortions and a number of passionate attitudes. After this seizure consciousness is recovered, and certain hallucinations and visceral disturbances, lasting often for a considerable time, may develop.

The patient may for days before the attack or series of attacks complain of malaise and digestive disturbances, such as anorexia or vomiting, or of circulatory symptoms, as for example vertigo or faintness, and she may be obviously depressed or excited. The hysterogenic zone (ovarian) becomes more sensitive, slight friction being at times able to cause the onset of a convulsion, and the hemianæsthesia, if present, becomes more intense and widespread. There may be muscular tremors or cramps, and even visual hallucinations of, for example, cats, rats, snakes, and other objectionable objects.

After a variable period, the definite aura, globus hystericus, begins as a severe (left) ovarian or iliac pain, which causes one of the following symptoms, or passes, in order, upwards to the epigastrium, causing nausea and vomiting: to the chest causing palpitation, to the neck causing a sense of constriction or of choking and sobbing, and finally to the head, giving rise to a feeling as if a nail were being hammered into the skull, clavus, together with dimness of vision and a hissing in the ears.

The hystero-epileptic fit differs in many essential respects from that of ordinary epilepsy. It may, for example, be diminished in severity or even stopped abruptly by pressure, or the application of electric currents over hysterogenic points, especially the ovarian. The fit begins by a loud groaning cry, or by a hissing inspiration, or several guttural noises. There is considerable agitation of the limbs, respiration ceases, and the patient falls down unconscious. Pallor of the face then gives place to redness, the neck swells, the face is distorted, the tongue is protruded, and the lips are covered with foam. Slow and stertorous respirations occur; there may be hiccough; the pupils are dilated and the eyes open and shut; or the lids are closed and tremulous, and resist opening, apparently owing to the presence of the corneal reflex. The body becomes rigid, in a condition of tetanic fixation. The clonic stage begins, after a longer or shorter period, with small rapid twitchings, which gradually increase in amplitude till they develop into large convulsive jerks. At the same time, clonic spasms of the facial muscles cause horrible grimaces. The fit, after one to five minutes, passes off, and the patient lies on her back completely paralysed. The face is congested and swollen, the respirations are stertorous, and the eyes are closed. This stage of resolution lasts from two to three minutes.

The hysterical portion of the seizure then begins as a series of contortions or large rhythmic movements. The patient places herself in strange attitudes, for example, resting on her head and heels, as if in a condition of tetanus (*l'arc de cercle*). The position assumed is usually one of opisthotonos, and more rarely of pleurosthotonos or emprosthotonos. The patient then bends backwards and forwards on her pelvis many times in succession (" salutation "), making loud whistling cries, or raises her feet in the air, resting her weight on her head and arms (" clownism "). These strange movements are readily performed by apparently weak girls. This stage of the attack lasts from five to ten minutes, and is followed by an emotional phase.

During this latter phase the patient gives expression by her countenance and attitudes to various rapidly changing emotions, which always occur in the same order in the same patient. She may, for example, exhibit in an extreme degree such passions as fear, hatred, erotism, and disgust. She may recall a whole scene of her past life, especially that part which is related to the original onset of her hysterical symptoms. As a rule, the mimicry is of two kinds—the happy and the sad. In the former, the patient imagines the existence of pleasant surroundings, music, and people clothed in red, which may be associated with or followed by the presence of erotic thoughts. In the latter she dreams of blood, murder, fire, or other alarming ideas. The attack may, as a rule, be stopped at any stage by the application of pressure in the ovarian region, and the patient may be then questioned about the particular emotion under which she is at the time labouring. The emotional stage is of variable duration, and usually lasts for several minutes.

Finally, consciousness is recovered, but a post-hystero-epileptic mental state may set in, and last for hours or days, or the whole seizure may recur again and again. The psychic derangement may take the form of hallucinations, of unpleasant objects, such as rats, crows, black cats, and vipers, or there may be visceral disturbances, with hiccough or borborygmi, or the patient may suffer from painful contractions of the limbs. Usually the attack is followed by a copious secretion of watery urine, and more rarely of vaginal mucus or saliva. As a rule, the patient remembers nothing whatever of the fit, but she may be able to recall the hallucinations to a greater or a lesser extent.

Numerous varieties of the hystero-epileptic seizure have been described by Richer, according as one or another stage of the fit is the more predominant. If the first stage only is well marked, *forme epileptoide*, the attack much resembles an ordinary epileptic fit, except in the points that there is no rise of temperature, and that the other stages may be observed to exist in a rudimentary condition. If the second predominates, *forme démoniaque*, the chief symptoms are strange attitudes, distortion of features, protrusion of the tongue, or extension of the first three fingers of the hand as if in the act of blessing. The third stage existing practically alone, *attaque d'extase*, can be reproduced by inhalation of ether. The patient under these circumstances strikes one, or it may be several, passionate attitudes. Either the second or the third stage may much resemble the attitudes frequently noticed in England in certain cases of chronic insanity. Finally, if the post-hystero-epileptic condition is the only one well marked, *attaque de délire,* the patient talks rapidly, prophesies, etc., the mental state usually, however, following a kind of epileptiform seizure. Again, cataleptic or somnambulistic phenomena may occur. These, as a rule, come on after the first or second period, and replace the third, or they may exist between two epileptiform seizures.

The more severe cases of hystero-epilepsy are apparently more common in France than elsewhere, though this condition is not necessarily peculiar to persons of any one race. The attacks of hystero-epilepsy probably bear a close relationship to the conditions occurring during religious exaltation, which have been described in all ages as the characteristics of the "prophetess," and to the various phenomena which have been ascribed to demoniacal possession. In other words, these conditions have in the past been considered to have their origin, in possession by either good or evil spirits.

It is probable that in Britain the majority of such cases, modified, of course, owing to racial characteristics, are to be found in the chronic wards of asylums. Whilst true cases of hystero-epilepsy rarely or never occur in these institutions, almost every symptom which has been described in the preceding account of the symptomatology of hysteria is more or less frequently to be met with in the insane. In view of the probable relationship between these conditions and hysteria, the fact that patients exhibiting these symptoms rarely develop any obvious amount of dementia, is of considerable significance.

In fact, the suggestion made earlier in this article, as an inference drawn from the description of the mental symptoms of hysteria, may be repeated with reference to the whole symptomatology. It is possible that hysteria and the various associated conditions which are met with in the insane may be ascribed to functional disturbances of the psychic processes, the remainder of the insanities (excluding the amentias), in which the psychic disturbances are usually or invariably associated with more or less neuronic degeneration and dementia, being placed in a separate group. This hypothesis, of course, does not preclude the frequent association of functional disorder of any part of the nervous system with cortical neuronic degeneration and true insanity, but is postulated in order to more clearly bring forward the definite relationship between hysteria and mental disease.

Diagnosis.—Owing to the frequent coexistence of emotional instability and organic disease of the nervous system, and to the occasional similarity of the symptoms of the latter to those occurring in hysteria, it is at times by no means easy to make a diagnosis between hysteria and organic disease. This difficulty is especially marked in the case of organic diseases with variable symptomatology, as for example disseminated sclerosis, particularly if the patient be young and of the female sex, for under these circumstances a certain amount of emotional instability will probably exist. In many of the cases of this class, however, careful examination will enable an obvious symptom of hysteria to be detected, as for example concentric contraction of the fields of vision, affection or even loss of colour vision, hysterogenic zones of hyperæsthesia, hemi-anæsthesia, involving the mucous membrane of the pharynx, and perhaps the special senses, or alternating recovery and relapse as regards the motor symptoms.

It is unnecessary to refer to the diagnosis of the mental symptoms, when these exist alone, as the only question which could arise would be a decision between hysteria and insanity, and the diagnosis made would depend on the possibility of home treatment or the necessity of restraint.

When physical symptoms coexist, their heterogeneous nature frequently enables a diagnosis to be made at once, as in the case of a girl of 16 years, who suffered from paraplegia without muscular wasting, from almost complete blindness of the right eye without loss of conjunctival sensation, and from frequent attacks of hæmoptysis.

If the case be more obscure, the following symptoms, which are characteristic of hysteria, are of especial importance in the diagnosis. The pains from which the patient suffers are altogether excessive in degree, they are liable to suddenly come on and as suddenly cease, and local signs of visceral disease are absent. The complaints of the patient concerning them are also, as a rule, abnormally persistent. Hysterogenic zones, existing at any rate as hyperæsthetic areas, are common. Anæsthesia is a very frequent symptom, occurring as a single or double hemianæsthesia, which involves the mucous membrane of the back of the pharynx, and often the special senses. The anæsthesia, when local, usually occupies a part of the body, as the patient knows it, quite apart from nerve distribution. Further details concerning this symptom will be found in an earlier paragraph. Limitation of one or both the visual fields is common, and affection of colour vision is by no means rare. Hysterical spasms and contractures are fairly common, and frequently the position of the limb is characteristic. These disorders may be fairly persistent, but it is by no means uncommon to find the spasm rapidly subside, or even come and go at intervals. Varieties of paralysis are fairly common, and occasionally, whilst co-ordination and muscular power are apparently normal when the patient is lying down, she is quite unable to stand or walk. The most common of the paralyses are hemiplegias, without affection of the face, and paraplegias. These are, as a rule, flaccid in character, and, being of cortical origin, they are associated with marked loss of muscular sense and tactile sensibility, which is limited by a circle above the joint affected with the paralysis. There is also neither a reaction of degeneration nor a loss of reflexes, and any wasting which exists is distributed generally over the paralysed parts. A further important point in the diagnosis of hysterical paralysis is the entire absence of effort to move the limb on the part of the patient. The limb hangs to the patient as if it did not belong to her, and if a leg, it is dragged after her. In aphonia, which is a particularly common variety of hysterical paralysis, the patient can cough loudly, but cannot speak beyond a whisper, and anæsthesia of the fauces is present.

In the case of functional cerebro-spinal motor and sensory affections, the diagnosis is beset with difficulties, and, unless definite hysterical symptoms coexist, it will in many cases be impossible, owing to the extreme difficulty of satisfactorily proving the absence of an organic cause for the symptoms.

The visceral symptoms of hysteria are, as a rule, of importance, as complicating sensory and motor symptoms, in which case they greatly increase the ease of diagnosis. This is particularly the case as regards throbbing of the large vessels without any coexisting valvular lesion, and abnormal irritability of the cutaneous vessels leading to readily induced artificial urticaria. Attacks resembling angina pectoris are not uncommon, and have been sufficiently referred to in the symptomatology. Spurious hæmoptysis is a valuable and fairly common symptom, as is also the excessive and violent hysterical cough. Amongst the digestive symptoms an abnormal secretion of gas in the intestines, accompanied, as a rule, by noisy eructations and borborygmi, is of considerable value in the diagnosis of certain cases, and it is probably worth while to draw attention to the frequency with which large quantities of pale watery urine are secreted.

With reference to the hysterical attacks, little need be said when they are of a purely emotional character, and particularly if, as is frequently the case, hysterogenic zones are present. When, however, a more or less marked epileptiform seizure occurs, some difficulty may exist as regards the diagnosis, especially if the whole of the attack is not seen. It is, however, uncommon for emotional disturbance to be entirely absent, and there is no rise of temperature. There may be loss of consciousness, but frequently the eyelids are closed and resist opening, apparently from persistence of the corneal reflex, and sometimes considerable swelling of the neck occurs. Usually, too, when the medical attendant arrives after the attack, he finds the patient struggling with the bystanders, and trying to kick or bite, a condition hardly likely to occur in true epilepsy.

To sum up, the diagnosis of hysteria can in many cases be made without much difficulty, owing to the frequent coexistence of emotional instability, and even definite hysterical attacks with heterogeneous symptoms, some of which are visceral and others sensory and motor. In the case of the latter the affection is usually one of a part of the body as an uneducated patient would describe it, rather than of a region presided over by a definite neuronic group; and it is by no means unusual to find the disability varying in degree from time to time. The history of the case is also of considerable importance, particularly with reference, in cases with localised symptoms, to an acute onset following a sudden shock, etc.; and according to the symptomatology present, the age and sex of the patient are often of great value.

Finally, it is necessary to add that there is hardly any disease of the nervous system which may not be simulated to some extent by hysteria, and that cases occasionally occur in which, after the use of all the resources of neurological science, it is practically impossible to make a certain diagnosis, and in which it is necessary to treat the patient as if she were suffering from serious organic disease of the nervous system. Fortunately, however, such cases are relatively rare, for usually some symptom characteristic of the organic diseases simulated is absent, or some symptom which could only be functional in origin, is present; therefore the number of such cases may almost be said to vary inversely with the knowledge, skill, and experience of the investigator.

Treatment.—Preventive.—In the preventive treatment of hysteria, the only etiological factor of importance, namely, the environment of the patient, should be under the control of the medical attendant. In many cases, as has been stated in the paragraph on etiology, the parents are almost the worst guardians who could well be chosen, and it may be desirable to remove the patient from the home surroundings, in order that the necessary régime for the development of healthy habits of living may be applied. Under the influence of regular hours, out-door occupation, and wholesome feeding, particularly if the mental education be properly adjusted to the physical, the inherited nervous instability has no incentive to exaggeration. Mental stability will, under these conditions, increase with age, and the patient may, to all intents and purposes, be cured of the tendency to hysterical manifestations by the time she arrives at maturity. Unfortunately, however, this preventive treatment can rarely be applied, and as a result the treatment usually needed is that of the developed disorder.

Remedial.—There are two most important essentials in the satisfactory general treatment of hysteria. These are the restoration of the general health, if, as is usually the case, it is impaired, and the necessity of gaining the complete confidence of the patient. As the case is one of disorder due to nervous instability, no method of treatment can have the slightest success unless it be believed in by the patient. Of all diseases and disorders, hysteria is the one to which the "faith cure" is especially applicable, and it is consequently from the treatment of this condition that the richest harvest of the quacks is derived. Whether the case be treated by the placebo method, by the use of nauseous anti-spasmodics (such as asafœtida and valerian), by the application of complicated procedures (such as the use of metals, magnets, or electricity, which are credited with an apparently miraculous power of, for example, transferring paralysis from one side of the body to the other), or by hypnotism—one object and one only is sought for, namely, a cure by suggestion to the trustful patient that this will result from the treatment adopted. Undoubtedly many of the methods successfully applied by eminent physicians savour strongly of charlatanism, but the fault, if fault it be, lies rather in the nature of the disorder than at the door of the physician. For the alleviation of such a protean disorder, in which many of the symptoms, both in their nature and in the manner in which they appear and disappear, hardly fall short of the miraculous, and in which the patient herself is frequently attracted rather than repelled by extraordinary, or at any rate unusual, methods—any variety of treatment, even one which antagonises artificial fraud against what is really a fraud of nature (though not a conscious one of the patient), is justifiable, so long as the end desired is obtained.

It is consequently frequently necessary for the physician to act the double deceiver—on the one hand, by pretending, as far as the patient herself is concerned, to believe in the serious nature of her complaint, which is very real to her; and, on the other, by suggesting that a cure will result from a certain method of treatment, when it really is due solely to the suggestion of recovery.

Whilst the methods just referred to are useful in a large number of cases, there are nevertheless many to which they are inapplicable. These are chiefly the cases in which it is necessary not to cure special symptoms in a patient who does not show obvious evidence of loss of control, but to stimulate the habit of self-control in patients who are obviously deficient in it. In such cases sympathy is frequently merely thrown away, and what is needed is a stimulant to self-control. Here either the physician or the friends must act on the principle of "standing no nonsense," and a cold douche when an attack of excitement occurs, or deprivation of a particular form of enjoyment if the patient has misbehaved herself, will frequently have a remarkable effect. These cases practically require the treatment meted out to the naughty children, of which they are a variety, in order that habits of self-control may be gradually developed.

In other cases, especially those in which the patient has, owing to her habits, become much run down in health, a modification of the treatment introduced by Weir Mitchell and Playfair will be found useful. The essential points in this method are isolation of the patient, to remove the injudicious influence of sympathising friends and relatives; forced feeding,

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to improve her general bodily, and, as a result, her mental health: and massage, to enable the excessive amount of food eaten to be assimilated. For this treatment it is naturally highly desirable that the patient should be removed from her home. She is placed in bed, and is put at first on milk diet. Massage is begun in two or three days, and faradism a week or so later, in order to stimulate muscular metabolism, and from about a quarter of an hour a day at first this treatment is increased in duration to as long as one and a half hours daily, at the expiration of a few weeks. As the duration of this treatment is lengthened, the milk, and, later on, milk and solid food, is increased in quantity; and according to the efficacy of the massage more and more food is taken, till enormous quantities are consumed and absorbed, and the patient increases markedly in weight. After each massage the patient should be wrapped up, and the room darkened in order that she may rest; and during this treatment it is highly desirable that both the doctor and the nurse should help and encourage her, all the time, however, so as not to cause any excitement, tactfully exercising the necessary moral control. The patient should, if emaciated, gain at least 2 lb. a week, otherwise the treatment should not be persevered with. If, on the other hand, the patient be stout, it is frequently desirable to diminish her weight, by rest in bed and low diet, before the treatment is commenced. The after-treatment consists in graduated exercise, and usually a more or less lengthy holiday before returning to her home.

It is unnecessary to refer here at length to the treatment of individual symptoms, as a suitable method will readily suggest itself in most cases. In many examples of paralysis, the result of judicious faradisation is striking, and in no variety is this more marked than in hysterical aphonia, where one or two external applications of a wire brush to the larynx usually result in a rapid cure. For the frequently coexisting dyspepsia the usual remedies are indicated, and are, as a rule, when combined with a suitable dietary, rapidly successful. In cases of long-standing contracture of the limbs, it may be necessary, under an anæsthetic, to break down adhesions, if they exist, or to perform tenotomy, the subsequent treatment consisting of massage and frequent passive movements of the limb, even if these be painful.

It may finally be added that, whilst many cases tax the resources of the physician to the utmost, the number of failures decreases *pari passu* with the increase in his skill, versatility, and tactfulness.

F. W. MOTT.

HYPOCHONDRIASIS.

UNDER the term of hypochondriasis is grouped a large number of cases, of which the important characteristic is perversion of the cerebral representation of organic sensation. As has already been stated, hysteria and hypochondriasis are, in this article, considered to be the same disorder, modified in symptomatology owing to differences in the age, sex, and temperament of the sufferers. Thus young women suffer from hysteria, or emotional instability, usually associated with physical disabilities, chiefly of psychic origin, of the ordinarily recognised "parts" of the body, especially the external. On the other hand, young men, and middle-aged or elderly persons of both sexes, but chiefly the male, suffer, owing to their being more prone to introspection of their symptoms, from hypochondriasis. By this term is connoted a disorder, of which the psychic symptoms are abnormal anxiety, nervousness, or distress, and the physical correlative is a disability, also chiefly of psychic origin, concerning the parts of the body of whose existence the patient is normally not obviously aware, namely, those concerned with the organic functions.

Relations.—As the varieties of hysteria grade insensibly into the large group of "moral" cases of insanity, which are not usually associated with dementia, so those of hypochondriasis imperceptibly pass into the ordinary (non-demented) varieties of visceral melancholia. A comparison, from the point of view of age, sex, and temperament, of the varieties of hysteria and hypochondriasis, with the corresponding types of true mental alienation, shows a striking coincidence, and in this supports the view that these are the important factors in the production of the special symptomatology occurring in the different patients.

Owing to the ignorance of the ordinary patient concerning the contents of his head, thorax, and abdomen, abnormally perceived sensations arising in these parts of his body are much more liable to give rise to morbid introspection than are the ordinary symptoms associated with simple mental instability. For example, a patient, with a pain or a curious sensation in the region of his diaphragm or in his head, or with frequent seminal emissions, when he knows that he has immorally indulged in normal or perverted sexual excess, is much more likely to suffer from extreme mental worry and anxiety as to what is really the matter with him, than is another who is excitable and unstable, and suffers from spasm or contracture of portions of an arm or leg, to whatsoever cause this may be due. The relationship between hypochondriasis and insanity has consequently been so universally admitted, that a new term, neurasthenia, has of late years been in use for a certain group of cases of the former, which for the purposes of treatment are conventionally considered *not* to be related to insanity, and the more marked cases of hypochondriasis have been handed over to the alienist, as if to bribe him not to claim the whole as belonging to his province.

It is one of the aims of the present article to draw especial attention to the corresponding analogues of hysteria which are seen in asylum practice, and, grouping together ordinary hysteria and hypochondriasis, to suggest that these terms probably connote a large group of cases of hereditary instability or purely functional disorder of the psychic regions of the cortex. In these cases the disorder is probably of a lower order in psychic development than is that in ordinary cases of mental alienation, in which the disease is associated with the higher psychic manifestations, and in which the final result is a more or less marked degree of higher neuronic degeneration and dementia. Owing to their tendency to introspection, cases of hypochondriasis are more liable to end in higher psychic disturbance, and even in true mental alienation in hereditarily disposed subjects, than are cases of hysteria. In asylum practice, however, it is probable that an equally large or even a larger number of cases of mental instability are under treatment, for, though violent attacks of excitement and loss of control are less likely to lead to mental enfeeblement, the patients suffering from them are more liable to require asylum régime, owing to their behaviour to other people, than are those whose attention and anxiety are concentrated on their own ailments, and who are consequently a nuisance rather than a dread to their friends and relatives. An interesting relationship exists in the onset of the more marked examples of the two conditions. Ecstatic religious excitement often begins with a sudden "conversion," after which the subject feels quite a different individual, and in the more severe forms of hypochondriasis the patient frequently states that the condition began suddenly with a feeling as if something had "given way" in, for example, the head. As an instance may be cited a case where the patient stated that she suddenly felt a snap in her head, after which she felt quite different. This snap immediately followed a prayer to God, in a moment of despair, that He would take her child from her, and was, she considered, due to the impious nature of the wish. These cases are especially intractable as regards treatment, owing to the existence of a fixed psychic explanation for the condition.

Symptoms.—In accordance with the definition given above, patients suffering from hypochondriasis usually refer their trouble to their head, digestive tract, or genital organs, though it is not rare to meet with cases who complain of their lungs, heart, or kidneys. A general type is also common in which the symptoms may be referred to all or any of the parts of the body, but especially those concerned in organic sensation.

In the majority of instances the disorder develops at or after the middle period of life, though it may occur at any age. It is more common in the male than the female sex, and this is especially the case in youthful subjects.

In youth and early middle age.—Whilst young women are more liable to suffer from emotional instability, young men suffer more frequently from hypochondriasis, though neither disorder is peculiar to either sex. In the latter cases the patient usually attributes his condition, either privately or in confidence to his medical attendant, to normal or perverted sexual excess. The basis of the symptoms is, as a rule, dyspeptic in nature, but the disorder is magnified by introspection until the patient shuns society and even believes that he is impotent. The patients are thin and pale, and have a dull eye and a haunted look. They are exceedingly miserable, and may even be suicidal, and they sleep badly. They are quite unable to concentrate their attention on their work, and may spend their whole time in studying their symptoms, and most of their money in purchasing various quack remedies. They have strange sensations or a feeling of weight and heaviness at the vertex of the head, and they complain in the most serious manner of the ordinary symptoms of dyspepsia, and give at times quite interesting theoretical explanations of the sensations from which they suffer. The prominent characteristic of the symptoms is the fact that they are sensations of various kinds rather than pains or tenderness, and frequently the patient's description of these and his explanation of them are so confused or so extraordinary, as in themselves to serve as a diagnosis. In one case a patient explained his solitary habits, on the ground that he was not in a position to keep a wife in luxury, and so avoided women lest they should fall in love with him. He suffered from a whole train of hypochondriacal symptoms, and these were eventually traced to the presence of the frænum, which he had convinced himself was an abnormality and necessarily made him impotent. When he was reassured on this point, he rapidly improved. These cases are, as a rule, readily cured, if the medical attendant gains the confidence of the patient, orders exercise, and treats the dyspepsia.

In the middle-aged and elderly.—In older people the symptoms are practically infinite, the description and explanation of them depending upon the education of the patient and upon the results of his study and introspection of his condition.

In cases of the general type, the organic sensations may, as a whole, be exaggerated or they may be dulled. An illustration of the former has been referred to under Hysteria. In another variety, the patient, who is usually of the male sex, is continually worrying about himself, and fearing that some function is not working in quite a normal manner. He looks upon himself as a "case," and studies in turn his pulse, his temperature, his tongue, and his bowels, as if he were the only person in whom they existed. He acts as if it would be little short of a miracle, at any rate in him, should they all act normally at the same time, even under his personal supervision. These cases, when of moderate severity, constitute the most common, and also the mildest, variety of the disorder, and they usually occur amongst ordinary male club-patients or hospital out-patients. Most of these patients suffer from a moderate amount of dyspepsia, and they never attempt the prescribed dietary treatment, but expect the medicine given them to at once cure them. A sharp purge may improve them for a day or two, but they soon drift into their ordinary condition, owing to the settled conviction that there is really something seriously the matter with them, and then they try either the same or another doctor.

In the cases where the organic sensations as a whole are dulled, the patient feels as if his whole body were dead, or as if he had no limbs, or no contents in his abdominal cavity or his head. He does not, as a rule, worry about this, except in so far that he either wants burying, as he considers he is dead, or he refuses food because he has no mechanism for its digestion. It is needless to say that the majority of the cases of this type are under asylum treatment.

In the cases where the seat of trouble is the head, the patient may complain in all seriousness that his brain is softened, and that he can feel its parts rolling about when he shakes his head, or may say that his brain is enlarged or shrunken. He describes the most extraordinary symptoms, and it only requires a step further for him to state that he has an electric machine, or some other object, inside it. This type of patient is always worrying about his brain, and is constantly trying different doctors and different treatments, and, owing to some fixed delusion, is frequently certified as insane, and sent to an asylum. In those cases where a group of *visceral sensations* are abnormally interpreted, the patients may constantly complain about their throats, and be firmly convinced that they suffer from serious organic disease, which is not diagnosed, solely owing to absence of skill on the part of the different specialists they consult. Others again complain of an obstruction in their œsophagus, or in their lower bowel, or of some serious disorder of some part of their intestinal tract. The descriptions given by the patients of the various symptoms from which they suffer are occasionally rather curious. They feel their food falling from their throat into their abdominal cavity, or they can recognise lumps of food circulating in their veins, or they, shortly after eating, cough their meal up, in which case it does them no good, and consequently might just as well not be eaten. Again they will state quite seriously that their sputum comes from their liver, that their blood does not circulate, or that they are in feeble health, because all their blood passes out in their urine (urates). In most of these cases the apparent absurdity is really due to ignorance of anatomy and physiology, and is often a carefully thought out explanation, which, if the patient is not to go elsewhere, should be treated with, at any rate, apparent seriousness.

The varieties in which the reproductive organs are considered to be at fault occur more frequently in women, and the uterus or the ovaries are the organs complained of. The genital cases in men usually occur, as has already been stated, in youth or early middle age.

Treatment.—All these cases are intractable and rarely curable; no method of treatment is satisfactory, and if they are well-to-do patients, their medical attendant more than earns his money, owing to the long visits they expect, and the tedious reiteration of their symptoms to which he is required to patiently listen. As will have been understood from the above description, only the more serious cases and also the poorer classes of patients pass into asylums, as the worst that can be said of them is, that they are a nuisance to every one with whom they come into contact, owing to the fact that they are constantly endeavouring to find excuses for detailing their symptoms to, and for drawing on the sympathy of, all who will listen to them.

F. W. MOTT.

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